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# VITREOUS IMPLANT FOR RETINAL DETACHMENT

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The well-known basic treatment of simple retinal detachment entails diathermy coagulation of the retinal tear(s) and the underlying area of choroid. The sub-retinal fluid is then evacuated through a perforation made in the sclera and choroid. This allows the retina to fall back into position. The ensuing inflammatory reaction causes choroido-retinal adhesions that seal the retinal tear(s). Anything preventing the retina contacting, or maintaining contact with, the choroid in the coagulation area will result in failure. Among procedures employed to maintain this contact are the various types of scleral 'resections' and the intra-vitreal injection of air, saline and even cerebrospinal fluid. Vitreous itself was first extensively used for this purpose by Shafer.[1] The intra-vitreal pressure after air or saline injection soon drops as the air or saline is absorbed. Since vitreous is not absorbed by the retina and has a colloidal viscous nature, it maintains its osmotic tension. It therefore has the advantage of maintaining a better intra-ocular pressure.

Shafer obtains the vitreous from eyes donated to the eye bank. It is collected by aspiration through a No. 18 BD needle, taking aseptic precautions. About 2 ml. of vitreous are obtainable by this method, and this is stored in glass vials at 4°C. It has been found suitable for use for up to 3 months ; 48 hours before operation the vitreous is cultured to ensure its sterility.

## TECHNIQUE

At operation the eye is first treated by diathermy to coagulate all retinal tears, and is then prepared for the vitreous implant as follows: A small incision (2 - 3 mm.) is made through the sclera, 8 - 9 mm. from the limbus, exposing the uvea in either the upper or lower temporal quadrants. Two opposing mattress sutures are placed in the sclera in readiness to seal the incision later. Drainage of the sub-retinal fluid is effected by scratching a small opening through the sclera in the detachment area to expose the choroid, which is then carefully perforated. The uvea in the prepared scleral opening is now punctured with a Graefe knife, and the vitreous is injected, using a No. 18 BD needle introduced through the opening and directed towards the centre of the eye. One mattress suture with a half-hitch is drawn by an assistant to seal the opening before the vitreous is injected. The implant is done slowly to raise the intra-ocular pressure a little above normal. This is maintained for a few minutes to allow all the sub-retinal fluid to drain. The tension is then reduced to about the upper limit of normal by relaxing the pressure on the syringe. The assistant maintains the tension on the half-tied suture to close the scleral wound as the needle is withdrawn. The second suture is then tied to make doubly sure that there is no leak.

This procedure can have its intricacies. The suture material must run easily through the tissue so that the incision is quickly closed as the needle is withdrawn, to prevent a great reflux of vitreous. Even so, with the suture well tied, the wound might not be completely sealed. A single half-hitch is better than a double half-hitch, i.e. the first stage of a surgical knot, since a knot might stick before the wound is properly closed. A single half-hitch, on the other hand, might slacken as the second hitch is being tied. Excessive zeal on the assistant's part can easily break the suture.

Moffatt[2] and Shapland[3] have modified Shafer's technique in the following respects: The perforation for draining the sub-retinal fluid is made by catholysis with a needle 0·5 mm. in diameter. The implantation of the vitreous is through a specially designed 18 BS gauge needle that has a stop 1 cm. from a special sharp cutting point.

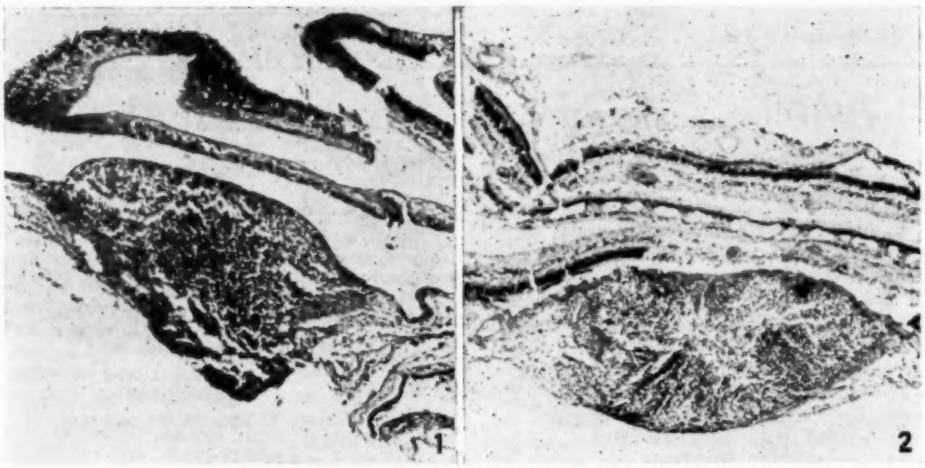
Their method of obtaining donor vitreous, however, is radically different. The donor eye, enucleated preferably within 6 hours of death, is placed in a penicillin-streptomycin suspension in liquid paraffin for 2 hours. Then, with all aseptic precautions, the eye is removed from the container, rinsed in Ringer solution and stored in sterile liquid paraffin after a culture has been taken from the limbus and attached bulbar conjunctiva. At the operation the stored eye is rinsed in sterile normal saline. The globe is perforated with a cautery, making an opening large enough to admit the nozzle of a 2 ml. record syringe. Then, with pressure on the globe and suction by the syringe, about 2 ml. of vitreous can be aspirated ; this is implanted.

Their experience has been that vitreous obtained from eyes prepared and stored in this fashion has been sterile. However, vitreous obtained in this manner is potentially dangerous, as will be described below.

The aspiration of more than 2 ml. of vitreous by a syringe is extremely difficult, and practically none can be so obtained from a child's eye. There is thus a large proportion wasted (the average vitreous body contains about 5·5 ml.) and the vitreous so obtained is very thin and liquid.

*Modified Technique*

For these reasons my method of collection is as follows: The stored eye, having been used for corneal grafting, is rinsed again in penicillin-streptomycin solution. All the remaining corneal, conjunctival and limbal tissue is removed. The iris is torn from its attachment. Loose pigment and debris is washed away by means of a pipette with the penicillin-streptomycin solution. The lens is then removed intracapsularly ; this is easily done in all cases and is good practice for the registrar. Another wash with



*Figs. 1 and 2.* Localized choroidal abscesses (haematoxylin and eosin × 45).

the antibiotic solution follows. The globe, held in a sterile gauze square, is inverted over a medicine glass and gently squeezed. The bulging hyaloid is cut with pointed scissors which are also used to cut the vitreous as it prolapses further. As soon as there is a suggestion that retina, uvea or tissue fluids might present and contaminate the vitreous, the process is stopped, but repeated, using a second medicine glass to try to collect more uncontaminated vitreous. In this way as much as 4 - 5 ml. of vitreous is obtained from one eye.

The vitreous in the glass is now poured into a 20 ml. dismantled syringe, which is held with its nozzle directed into the small sterile bottle in which the vitreous is to be stored. The plunger is now inserted into the syringe, and with a little force the vitreous suddenly passes through the nozzle. The bottle is sealed with a rubber bung and a metallic screw-cap, and is also covered with a sterile fingerstall. Forty-eight-hour cultures are taken before use.

The vitreous so obtained and stored is more viscid than that obtained by the other methods described. Furthermore, it has been, as it were, converted from a gel to a sol phase and now can pass through a No. 26 BSG needle. Thus the use of thicker needles and the preparatory scleral incision and mattress sutures are unnecessary. The Tolia cannulated needle-knife (Grieshaber) can be used very effectively. It is extremely fine with a really sharp cutting point. The recipient eye can be pierced extremely obliquely through the sclera and ciliary body, so that the puncture becomes self-sealing after removal of the needle. The extra-sharp point and cutting edge is specially important when perforating the ciliary body, for in a soft eye (the sub-retinal fluid has already been tapped) a not-so-sharp needle is more likely to produce tears and more damage than desirable.

As mentioned above, the vitreous can now pass through a No. 26 BSG needle. However, before the actual implant is done, it is advisable to 'test-inject' all the vitreous through such a needle. Small fragments of tissue or abnormal coagula of vitreous can thus be removed. Naturally, several needles must be available for this procedure.

On one occasion, after the vitreous had been collected, the remains of the donor eye were examined, and 6 small whitish nodules, miliary in size, were noted in the choroid. The other eye from the same donor showed 4 similar features. From the macroscopic appearance these nodules were thought to be early secondary tumour growths. The cause of death of the donor, however, was recorded as cerebral thrombosis—haemorrhage. The patient had been unconscious for several days before death. No autopsy had been done. Culture of the

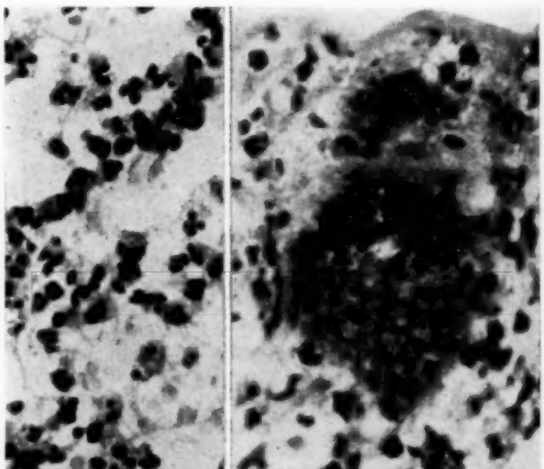


*Fig. 3.* Inflammatory-cell infiltrate at the edge of an abscess. Note the large number of polymorphonuclear cells (haematoxylin and eosin × 600).
*Fig. 4.* A mass of micro-organisms, probably cocci, in one abscess (haematoxylin and eosin × 750).

vitreous showed it was not sterile, and it was discarded. Unfortunately, no note was made of the organism, for the subsequent histological examination showed that the small nodules were early abscesses. Dr. J. C. Kaufmann of the South African Institute for Medical Research reported as follows:

'Sections from representative nodules show small localized acute abscesses in the choroid (Figs. 1 and 2). In addition to some products of cell destruction, these consist of many polymorphonuclear cells and some other inflammatory cells, chiefly mononuclear cells and histiocytes (Fig. 3). At one level in one of the abscesses just beneath Bruch's membrane there is a rounded mass of micro-organisms (Fig. 4). These are rounded in shape, vary a little in size, are very much smaller than 2 $\mu$, and stain blue-red by Wright and Craighead's method. They thus appear to be cocci and not toxoplasmata. Adjacent vessels are partly blocked by, and their walls are infiltrated with, polymorphonuclear cells, but no septic emboli are visible. In one part inflammatory cells extend deep into the retina as far as the ganglion-cell layer. Choroidal vessels show some hyaline arteriosclerosis and there is also some cystoid degeneration of the retina (Fig. 5).'

*Fig. 5.* Choroid and retina. Note the retinal vessel containing polymorphonuclear cells, hyaline arteriosclerosis of the choroidal vessels, and cystoid degeneration of the retina (haematoxylin and eosin × 240).

These incipient abscesses are therefore most probably a result of a slow dying process, with a bacterial invasion of the blood stream. The method of vitreous collection as described by Moffat[2] and Shapland[3] can therefore be a hazardous procedure.

## COMMENT

The vitreous-implant operation has usually been a last resort. Shafer pleads that it should be used earlier than at a third or fourth operation. The simplicity of the method of injection described above would allow this to be almost a routine procedure at the first operation (if available) whenever ophthalmoscopic examination shows the retina has not flattened out completely. This problem of availability should stimulate research into the feasibility of using animal vitreous in human beings.*

There can be no doubt that a vitreous implant at the first operation accounts for the successful outcome in the following case:

The patient was a World War I casualty. One eye had been enucleated. An intra-ocular foreign body and several others had been removed from the other eye. A traumatic cataract subsequently progressed, and in 1950 an extra-capsular cataract extraction was done. A dense membrane remained, but there was a small clear gap that enabled the patient to get about and read. Nearly 10 years later his vision suddenly failed. A retinal detachment was suspected, but since it was impossible to see any details, a capsulectomy was done which made it possible to confirm that the retina was severely ballooned and completely detached, at which stage the patient was referred to me.

A mesh of holes was found in the peripheral retina between 5 and 7 o'clock. Two small holes were also noted between 12 and 1 o'clock. At operation, the area of the retinal scars could not be coagulated with the diathermy, since the ballooning was too high. Thermo-couple electrodes of Cuepper were used and a scleral temperature of even 90°C. was tried. Small holes exposing the choroid were then cut in the sclera at about the equator in the 2 lower and upper temporal quadrants. The exposed uvea was touched with a heated Wadsworth cautery and the coagulated tissue then perforated. The lower quadrants only were perforated, the eye becoming so soft it was felt that to perforate the upper temporal quadrant might result in additional damage to the retina. The Tolia needle was then introduced extremely obliquely in the right nasal quadrant 3 mm. from the limbus. (In patients who have a lens the point of entry is 5 mm. from the limbus.) As the vitreous was implanted the retina was observed to flatten out. The advisability of applying diathermy now was considered, but in view of the amount already applied to the sclera it was decided not to manipulate the eye further.

The following day the patient reported his vision had returned. Some days later light coagulation was used to seal the area of the tears, and 2 weeks later still a small scleral buckling operation was done in the lower outer quadrant where retinal and sub-retinal striae were visible, apparently related to the course of the intra-ocular foreign body of World War I. Follow-up shows that the cure has been maintained.

## SUMMARY

A technique of collecting vitreous and a simplified method of implantation is described. The advantage over other methods is discussed. A preterminal bacteraemia might produce incipient inflammatory foci within the eye and infect the vitreous. One cured case with an extremely poor pre-operative prognosis is described.

I am indebted to Mr. M. Ulrich of the Photographic Unit, SAIMR, for the micro-photographs.

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* Preliminary animal experiments suggest that animal vitreous contains too much protein and may be antigenic. A search of the literature has revealed that it has been tried in humans, but the results were not conclusive.

# THE INFLUENCE OF ULTRAVIOLET IRRADIATION OF THE SKIN ON THE CIRCULATING BLOOD LIPIDS IN MAN*

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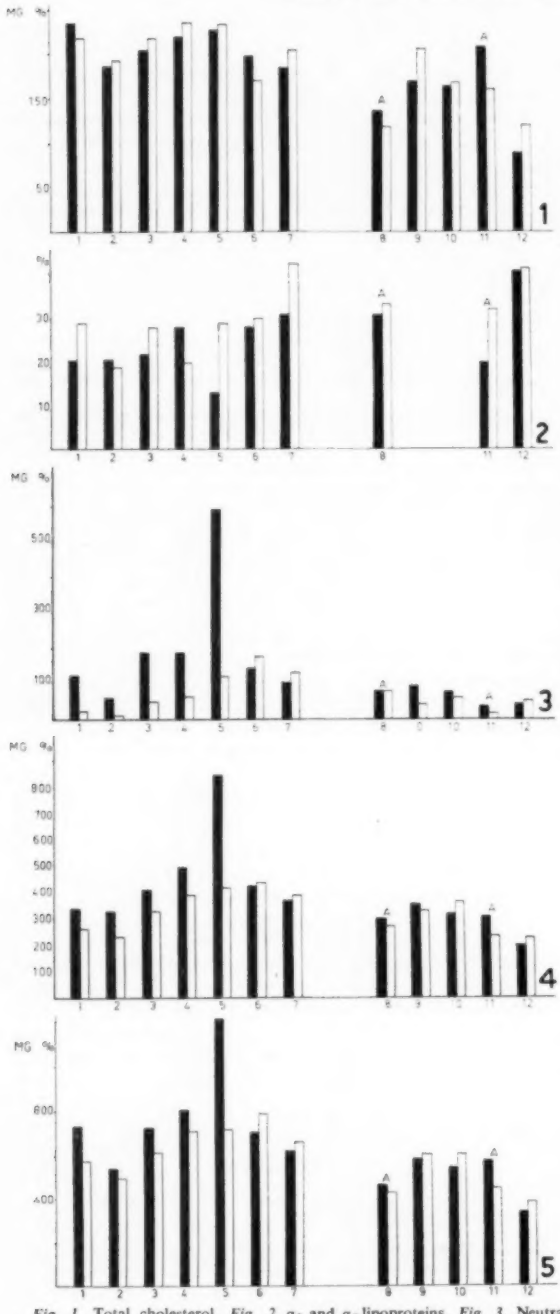

*Fig. 1.* Total cholesterol. *Fig. 2.* $\alpha_1$ and $\alpha_2$ lipoproteins. *Fig. 3.* Neutral fats. *Fig. 4.* Total fatty acids. *Fig. 5.* Total lipids. [In Figs. 1 - 5, numbers 1 - 7 represent the White patients, numbers 8 - 12 the Bantu (A=albino). The black columns represent estimations before irradiation, the white after irradiation.]

In the past the suggestion has often been made that sunlight is a precipitating factor in the pathogenesis of atherosclerosis.

The aim of this paper was to collect data on the influence of ultraviolet irradiation on the blood lipids.

## MATERIAL AND METHODS

Hospitalized patients who were out of direct sunlight for at least 1 week were exposed to increasing amounts of ultraviolet irradiation for 1 week. Blood lipids were estimated directly before and directly after completion of the course of irradiation. The patients were kept on the ordinary hospital diet. Twelve patients suffering from chronic skin diseases (ulcus cruris, neurodermatitis, acne vulgaris, and plantar warts) were used in these experiments. Patients 1 - 7 were White and 8 - 12 were Bantu. Patients 8 and 11 were albino Bantus. In the case of the Whites and albinos, irradiation with a high-pressure mercury lamp (Hanau) was started on the chest and back for 1 minute and increased by half a minute daily for 7 days, at a distance of 24 inches. The other Bantus were initially exposed for 2 minutes with a daily increase of 1 minute.

## RESULTS

The results are shown in Figs. 1 - 5 and Table I. The fluctuations before and after irradiation were insignificant in the following estimations which are not shown in the figures: $\alpha_1$ and $\alpha_2$ cholesterol, $\beta$ lipoproteins, $\beta$ cholesterol, phospholipids, free cholesterol, cholesterol esters, and percentage esters of the total.

## COMMENT

In the case of total cholesterol the changes were also insignificant, with no constant tendency to either a decrease or an increase (Fig. 1). The $\alpha_1$ and $\alpha_2$ lipoproteins showed a tendency to increase after irradiation in 8 out of 10 patients (Fig. 2). A very marked decrease after irradiation was noted for the neutral fats in 7 of the 12 patients. In 2 there was a slight increase, and in the other 3 the fluctuations were minimal (Fig. 3). A similar tendency to decrease after irradiation was also noted, but to a lesser degree, for the total fatty acids and total lipids (Figs. 4 and 5). The cholesterol-phospholipid ratio was slightly increased in nearly all the patients (Table I).

TABLE I. CHOLESTEROL-PHOSPHOLIPID RATIO

| Patient | *Before irradiation* | *After irradiation* |
|---|---|---|
| 1 | 0·93 | 1·03 |
| 2 | 0·97 | 0·97 |
| 3 | 0·95 | 0·96 |
| 4 | 0·79 | 0·84 |
| 5 | 0·82 | 1·00 |
| 6 | 0·82 | 0·73 |
| 7 | 0·81 | 0·98 |
| 8 | 0·78 | 0·72 |
| 9 | 0·78 | 0·93 |
| 10 | 0·71 | 0·80 |
| 11 | 1·00 | 0·89 |
| 12 | 0·73 | 0·80 |

According to these experiments, ultraviolet irradiation seems to decrease the neutral-fat values of the blood, while no influence was noted on the cholesterol values. There also seems to be an increase of the $\alpha_1$ and $\alpha_2$ lipoproteins.

At present the significance of these findings remains obscure.

We should like to thank Dr. A. T. Neser of the South African Institute for Medical Research, Bloemfontein, for his assistance with the chemical estimations.

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## TOXAEMIA OF PREGNANCY

Toxaemia of pregnancy is a complicated problem which is of interest to scientists working in different disciplines, e.g. pathology, physiology, biochemistry, and many others. The term toxaemia has not been acceptable to many workers. It has been condemned as giving a spurious sense of unity to the various obscure complications in pregnancy and implying that they are caused by the action of a chemical substance or toxin.[1] One fact that is important in the natural history of pre-eclamptic toxaemia is the dramatic subsidence of symptoms after parturition. Parturition is accompanied by a disappearance of the causative agent of the hypertension and other features. It has long been thought that the placenta is a possible source of a toxin affecting the mother.

A tremendous amount of work has been done to solve the problem of the aetiology of the toxaemias of pregnancy, and many theories and facts are available from workers in many different parts of the world. While many significant changes have been demonstrated in the various organs, such as the liver and the kidneys, it has not been disproved that there is an association between placental changes and toxaemias of pregnancy.[2] One hypothesis based on experimental work states that an absolute or relative insufficient blood supply to the placenta is an important factor in the genesis of toxaemia.[3]

Vascular lesions are significant in eclampsia. Characteristic changes are found in the liver and the kidneys. In the placenta and other organs special changes have been described, different workers laying emphasis on particular observations they have made. The endocrine glands have been studied in detail, and some regard the rôle of the adrenal cortex of special importance. The foetus is not the cause of pre-eclampsia and eclampsia, since pre-eclampsia is many times more common in hydatidiform mole than in normal pregnancy, and eclampsia has occurred many times with moles. The chorionic villi of the placenta show syncitial degeneration in pre-eclampsia and eclampsia, but in hydatidiform mole there is proliferation of both syncitial and cellular layers.

It has been deduced that abnormal function of the maternal placenta appears to be the cause of pre-eclampsia and eclampsia, and that some alteration in the cells might be the stimulus for liberation of a factor causing maternal hypertension by chemical means.[4]

The hypertension that accompanies toxaemia of pregnancy has been relieved in 64 of 67 patients with acute toxaemia by postpartum uterine curettage. This observation has been made by Hunter[5] who reported last year the discovery of hysterotonin as a *pressor* substance in toxaemic mothers. The highest concentration of this substance was found in decidual tissue, and lesser amounts in the amniotic fluid and blood plasma. He and his colleagues followed blood-pressure changes after removal of 8 - 20 grammes of decidual tissue from each patient. In three patients with essential hypertension and no superimposed toxaemic hypertension, there was no response to curettage. The 64 patients whose blood pressure returned to normal had mild or severe pre-eclampsia. These findings are of great interest and importance, and further reports are awaited for confirmation from other workers.

The toxaemias of pregnancy are still major complications of human reproduction. The cause of pre-eclampsia remains to be established, and there are no specific therapeutic agents for its prevention or cure. The antihypertensive drugs are merely palliative agents, and although the blood pressure can usually be controlled by such drugs, the underlying disease process is unaltered. It is to be hoped that in the not too distant future a better understanding of the cause of the disease will be forthcoming, so that more rational therapy can then be attempted than has been possible up to the present time.

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## NARKOSETEGNIEK VIR MEDIESE STUDENTE

Met die verskyning van *Narkosetegniek vir Mediese Studente*[1] deur Gordon Ostlere en Roger Bryce-Smith, soos vertaal deur J. A. Pretorius en A. B. Bull, het daar weer eens 'n toevoeging gekom tot ons nog skrale voorraad van mediese hand- en leerboeke in Afrikaans. As sodanig wil ons die publikasie van hierdie boekie verwelkom. Die feit dat ons heel onlangs die verskrywing van die *Aptekerswoordeboek* in Afrikaans kon aankondig, en nie so lank gelede nie *Chirurgiese Ondersoekingmetodes* deur proff. F. M. Saint en J. H. Louw, toon aan dat die stroompie van Afrikaanse geneeskundige publikasies, alhoewel dit nog nie sterk vloei nie, tog daadwerklik aan die loop bly.

Een van die baie opvallende feite wat betref vordering met die Afrikaanse geneeskundige vaktaal gedurende die afgelope paar jaar, is die feit dat daar sulke groot vooruitgang gemaak is. Dit is bevredigend om 'n geneesheer oor enige moontlike tegniese onderwerp in die medisyne in vlot, vloeiende, idiomatiese en korrekte Afrikaans te hoor praat. Hierdie feit in sigself weerspieël 'n kulturele bydrae tot ons algemene kultuurstand in hierdie land wat van geen geringe betekenis is nie.

Die vertalers van *Narkosetegniek vir Mediese Studente* bied die poging aan in die hoop dat Afrikaanssprekende studente en kollegas die aanbieding van so 'n welbekende boekie nuttig en aanneemlik sal vind. Ons wil daarby

spesifiek die hoop uitspreek dat die boekie as grondslag vir die onderwys van studente in die narkoseleer gebruik sal word by altwee geneeskundige skole waar Afrikaans as onderrigmedium geld, en ook elders waar Afrikaanse studente en kollegas leer en werk.

Die vertalers besef dat die Afrikaanse vaktaal nog grotendeels in sy vormingstadiums is; daarom het hulle geen dogmatiese opvattings oor die aard en kwaliteit van die woorde en terme wat hulle gebruik nie. Hulle spreek dan ook die hoop uit dat die boekie, wat die terminologie betref, as *'n basis van bespreking* kan dien en stimulerend mag inwerk op andere wat geskrifte oor die narkoseleer in Afrikaans wil publiseer of bestudeer. Opbouende kritiek en konstruktiewe wenke sal dus hartlik verwelkom word.

Soos kenners van die vak wat bekend is met die boekie sal weet, handel dit hoofsaaklik oor een aspek van die narkose, naamlik, die toediening van algemene narkose. Hierdie vorm van narkose is egter die vorm wat meestal in die praktyk gebruik word. Dit is ook die vorm van narkose wat by die grootste verskeidenheid van chirurgiese tegnieke aangepas kan word. Daar is dus 'n groot aantal spesialisnarkotiseurs en algemene praktisynnarkotiseurs wat in die verskyning van hierdie boekie behoort belang te stel.

Oor die algemeen is die Afrikaans wat deur die vertalers gebruik is vlot, idiomaties en waardig, en hulle moet met hierdie poging gelukgewens word. Mag ons die hoop uitspreek dat baie ander kollegas in hul voetspore sal volg, wat betref die publikasie van vertalings sowel as van oorspronklike werk.

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# THYROID TREATMENT OF ESSENTIAL HYPERTENSION—THE IMPORTANCE OF EARLY DIAGNOSIS*

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In 2 series of cases of hypertension treated with desiccated thyroid[1,2] it was shown, firstly, that patients with renal hypertension failed to respond and, secondly, that the success achieved in the majority of patients was only partial, the blood pressure in these falling to levels intermediate between the original readings and the norm (group 2, Table I).

It is not difficult to account for these results. The explanation lies in the now accepted fact that hypertension of long standing, whatever its cause, produces narrowing of the renal arteries and arterioles. The resulting ischaemia then sets in motion the mechanism of renal hypertension.[3-8] It follows that the majority of cases of hypertension which are labelled 'essential' are in reality of mixed aetiology, 2 distinct mechanisms being responsible for the elevation of the blood pressure. Of these only 1 responds to thyroid treatment.

TABLE I. RESPONSE OF HYPERTENSIVE PATIENTS TO THYROID TREATMENT

| *Group** | *Blood pressure* | *No. of patients* | *% of total* |
|---|---|---|---|
| 1 | Fall to normal .. .. | 46 | 14 |
| 2 | Fall to intermediate levels | 183 | 55 |
| 3 | No fall .. .. .. | 105 | 31 |
| Total | | 334 | 100 |

* In the paper in which this table first appeared,[2] it was suggested that group 1 consisted of patients with 'pure' essential hypertension, group 2 of patients with mixed essential and renal hypertension, and group 3 of patients with predominantly renal hypertension.

If, then, we are to obtain more decisive results, it is obvious that we must begin treatment before renal hypertension supervenes. With this object in view a controlled experiment with patients whose pressures were only slightly raised was undertaken. In 12 consecutive experiments on 6 patients, the expected result was obtained.

Before giving the clinical details of these experiments, I propose to present the 2 cases which led me to conclude that such an experiment might prove helpful.

CASE 1

Mr. M.J.W., aged 26 years, married, a clerk.

*History*

*Complaint:* inclined to put on weight easily.
*Symptoms:* none.
*Family history:* mother obese.

*Progress (Fig. 1)*

| *Date* | *Blood pressure (mm. Hg)* | *Pulse rate* | *Wt. (lb.)* | *Thyroid (gr.) (daily dose)* |
|---|---|---|---|---|
| 1953 | | | | |
| 3 June .. .. | 140/90 | 76 | 174 | None. Diet |
| 3 July .. .. | 140/90 | 76 | 173 | 1 |
| 10 July .. .. | 140/90 | 80 | 173 | 2 |
| 24 July .. .. | 140/90 | 80 | 172 | 2 |
| 21 August .. | 128/80 | 80 | 170 | 3 |
| 4 September .. | 124/80 | 70 | 167 | 3 |
| 25 September .. | 126/72 | 68 | 164 | 3 |
| 23 October .. | 136/70 | 64 | 160 | 3 |
| 28 November .. | 130/70 | 72 | 157 | 3 |
| 1954 | | | | |
| 8 January .. | 124/70 | 72 | 156 | None |
| 5 February .. | 124/76 | 60 | 157 | None |
| 29 March .. | 132/94 | 62 | 161 | 3 |
| 30 April .. .. | 132/80 | 78 | 164 | 3 |
| 28 May .. .. | 128/74 | 68 | 163 | 3 |
| 25 June .. .. | 136/82 | 82 | 163 | 3 |
| 23 July .. .. | 128/80 | 72 | 166 | 4 |
| 26 August .. | 128/74 | 74 | 166 | 4 |
| 1 October .. | 126/64 | 66 | 166 | 4 |

*Comment*

Usually, the physician does not concern himself with blood pressures at this level. I did here, because I thought that a

* This paper — certain clinical details excepted — was read at the 6th International Congress of Internal Medicine held at Basle, Switzerland, 24-27 August 1960.

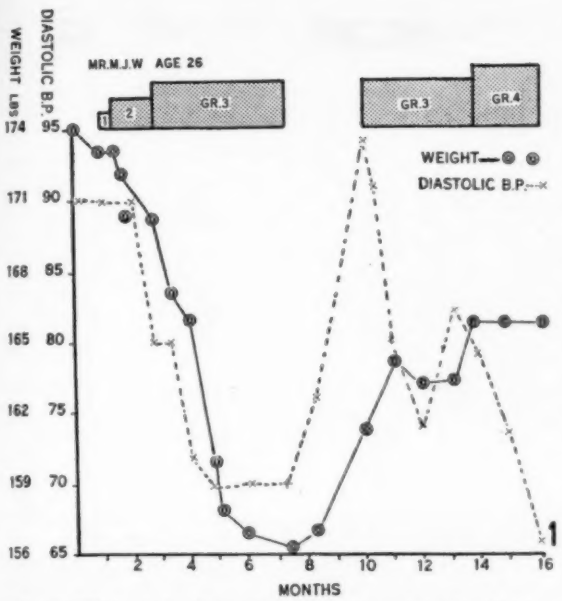



*Fig. 1.* Effects of thyroid on diastolic blood pressure and weight in early essential hypertension (case 1).

correlation between weight and blood pressure might prove of interest, particularly since some observers, such as Fishberg,[6] had stated that the fall in blood pressure in patients receiving thyroid is probably the result of weight loss. As will be seen from this record, the correlation is a very rough one. On 29 March 1954, with his blood pressure at 132/94 mm.Hg, the patient's weight was 161 lb. Six months later, while on thyroid, gr. 3 and later gr. 4 daily, he weighed 166 lb. His blood pressure, however, had fallen to 126/64 mm.Hg.

The most likely explanation of the relationship between obesity and hypertension is that both are manifestations of thyroid insufficiency.

The following details are worthy of note:

1. The remarkable consistency of the first 4 readings. Such readings, contrary to what was expected, were a feature of most of the cases diagnosed as early essential hypertension.
2. The disproportionate falls in the systolic and diastolic blood pressures. If we take 140/90 mm.Hg as the base line, then the maximum fall in the systolic pressure was from 140 to 124 mm.Hg, i.e. 16 mm.Hg, while the maximum fall in the diastolic pressure was from 90 to 64 mm.Hg, i.e. 24 mm.Hg.
3. The elevation of the blood pressure on stopping thyroid and its fall on resuming it.
4. The fall of the pulse rate on thyroid. This has been a common observation in hypertensive patients on this treatment. Note that towards the end of the experiment this patient was receiving a daily dose of 4 gr. of thyroid.

### CASE 2

Mr. F.W.R., aged 47 years, married, a journalist.

*History*

*Complaints*—tiredness, discomfort in chest on walking, overweight.

*Family history*—not relevant.

*Progress*

| *Date* | *Blood pressure (mm. Hg)* | *Pulse rate* | *Thyroid (gr.) (daily dose)* |
|---|---|---|---|
| February 1956 .. .. | 164/114 | 60 | 1 |
| February 1957 .. .. | 148/104 | 58 | 2 |
| March 1957 .. .. | 128/90 | 80 | 2 |
| March 1958 .. .. | 144/94 | 68 | 2 |

*Comment*

This patient's weight, unfortunately, was not recorded. As will be noted, with 1 exception the readings were at yearly intervals. In March 1957 he stated that he was feeling particularly well. The chest discomfort left him shortly after he began taking thyroid.

These 2 cases, and more especially the first, suggested the possibility of treating essential hypertension at a very much earlier stage than is customary. They also suggested the feasibility of an adequately controlled therapeutic trial.

In the past such trials have not carried conviction because of our inability to neutralize the numerous variables normally influencing the blood pressure. I refer to such variables as the emotional state of the patient between readings and his emotional state at the time the reading is taken; the amount of physical exertion between and just before readings; the degree of narrowing of the renal arterioles and hence the extent to which the renal factor is responsible for raising the blood pressure; and finally, the rate at which the renal component of the hypertension is progressing.

Though such trials are unlikely to prove helpful in patients with established essential hypertension, they might provide reliable results in the early stages of the disorder. Firstly, because it would be unlikely that these cases would be complicated by renal hypertension and, secondly, because the responses to emotion, physical exertion and other factors would be so small as to be conveniently disregarded.

Such a trial was undertaken in a small series of patients who, I felt, could be diagnosed as having early essential hypertension. The results in 6 cases are given below. Two of the patients did not realize that it was their blood pressures in which I was primarily interested. I provided them with the thyroid and with the dummy tablets.

### CASE 3

Mr. M.D.J., aged 32 years, married, a factory foreman.

*History*

*Complaints*—tiredness, frequent attacks of chest discomfort, overweight.

*Family history*—not relevant.

*Progress*

| *Date* | *Blood pressure (mm. Hg)* | *Pulse rate* | *Wt. (lb.)* | *Treatment (daily)* |
|---|---|---|---|---|
| 29 May 1953 | 140/96 | 88 | 204 | Diet only |
| 1 April 1957 | 142/94 | 90 | 206 | Diet only |
| 29 Oct. 1958 | 140/100 | 88 | 220 | Thyroid, gr. 2 |
| 12 Nov. 1958 | 130/90 | 80 | 216 | Placebo |
| 26 Nov. 1958 | 140/100 | 80 | 213 | Thyroid, gr. 2 |
| 10 Dec. 1958 | 134/86 | 80 | 213 | Placebo |
| 23 Dec. 1958 | 138/96 | 92 | 210 | Thyroid, gr. 2 |
| 11 June 1959 | 132/88 | 72 | 210 | Thyroid, gr. 2 |

*Subjective Improvement*

Definite. After 6 months on thyroid, gr. 2 daily, he stated that he no longer felt tired, that he was more energetic and that the chest discomfort had disappeared. Note the fall in pulse rate.

### CASE 4

Mr. B.E.R., aged 35 years, married, a draughtsman.

*History*

*Complaints*—heavy feeling over left chest for past 2 weeks, giddy spell for 4 days.

*Past history*—1946, blood pressure queried by insurance company; 1953, blood pressure again queried by insurance company.

*Family history*—mother hypertensive (blood pressure 260/140 mm.Hg).

*Progress*

| Date 1958 | Blood pressure (mm. Hg) | Pulse rate | Wt. (lb.) | Treatment (daily) |
|---|---|---|---|---|
| 29 Oct. | 156/100 | 68 | 196 | Thyroid, gr. 2 |
| 12 Nov. | 146/92 | 68 | 196 | Placebo |
| 26 Nov. | 150/100 | 76 | 196 | Thyroid, gr. 2 |
| 10 Dec. | 146/88 | 80 | 195 | Placebo |
| 23 Dec. | 156/100 | 64 | 194 | Thyroid, gr. 2 |
| 1959 | | | | |
| 23 March | 146/90 | 70 | 187 | Thyroid, gr. 3 |
| 18 May | 144/92 | 76 | 187 | Thyroid, gr. 3 |

*Subjective Improvement*

Definite. No more giddy spells. Occasional chest discomfort, but of short duration.

CASE 5

Mr. A.G.M., aged 38 years, married, a draughtsman.

*History*

*Complaints*—drowsy, cannot concentrate, overweight.
*Family history*—mother grossly overweight, mental symptoms.

*Progress* (*Fig.* 2)

| Date 1958 | Blood pressure (mm. Hg) | Pulse rate | Wt. (lb.) | Treatment (daily) |
|---|---|---|---|---|
| 28 July | 154/96 | 96 | 215 | Thyroid, gr. 1 |
| 11 Aug. | 134/92 | 76 | 215 | Thyroid, gr. 2 |
| 25 Aug. | 136/96 | 76 | 207 | Thyroid, gr. 3 |
| 9 Sept. | 126/88 | 84 | 210 | Thyroid, gr. 3 |
| 7 Oct. | 130/90 | 74 | 205 | None |
| 21 Oct. | 148/102 | 64 | 211 | Thyroid, gr. 2 |
| 4 Nov. | 130/86 | 68 | 209 | Placebo |
| 18 Nov. | 140/98 | 72 | 209 | Thyroid, gr. 2 |
| 2 Dec. | 136/86 | 86 | 212 | Placebo |
| 15 Dec. | 150/100 | 72 | 210 | Thyroid, gr. 3 |
| 25 Dec. | 136/90 | 78 | 208 | Thyroid, gr. 3 |
| 1959 | | | | |
| 24 April | 134/94 | 70 | 214 | Thyroid, gr. 2 |

*Subjective Improvement*
Uncertain.

CASE 6

Mr. I.H.M., aged 34 years, married, an accountant.

*History*

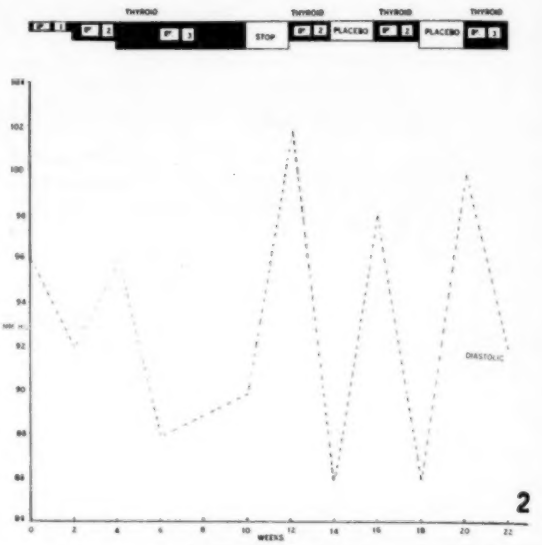
*Complaints*—tiredness, tendency to put on weight easily.
*Family history*—father and mother both have high blood pressure.

*Progress* (*Fig.* 3)

| Date 1959 | Blood pressure (mm. Hg) | Pulse rate | Wt. (lb.) | Treatment (daily) |
|---|---|---|---|---|
| 21 Jan. | 140/94 | 70 | 200 | Thyroid, gr. 2 |
| 5 Feb. | 128/84 | 72 | 200 | Placebo |
| 19 Feb. | 146/100 | 66 | 200 | Thyroid, gr. 3 |
| 5 March | 134/86 | 74 | 201 | Placebo |
| 19 March | 144/100 | 72 | 199 | Thyroid, gr. 3 |
| 16 May | 132/80 | 88 | 196 | Thyroid, gr. 2 |

*Subjective Improvement*

Definite. Less tired. At last visit stated he was feeling tired again. This was considered to be from overdosage in view of the pulse rate, and the dose was changed to gr. 2 daily.



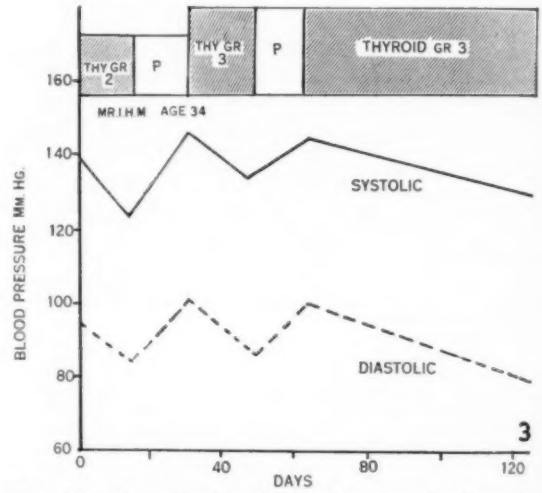




*Fig. 2.* Effect of thyroid and of placebo on the diastolic blood pressure in early essential hypertension (case 5).
*Fig. 3.* Effect of thyroid and of placebo on the blood pressure in early essential hypertension (case 6).

CASE 7

Mr. C.E.S., aged 25 years, single, a draughtsman.

*History*

*Complaints*—tendency to put on weight easily.
*Family history*—grandmother had hypertension.

*Progress*

| Date 1959 | Blood pressure (mm. Hg) | Pulse rate | Wt. (lb.) | Treatment (daily) |
|---|---|---|---|---|
| 18 May | 146/100 | 72 | 180 | Thyroid, gr. 3 |
| 2 June | 146/88 | 84 | — | Placebo |
| 16 June | 146/104 | 82 | — | Thyroid, gr. 3 |
| 30 June | 144/82 | 76 | — | Placebo |
| 14 July | 142/104 | 72 | — | Thyroid, gr. 3 |
| 10 Sept. | 130/90 | 76 | 172 | Thyroid, gr. 3 |

CASE 8

Mr. D.N.C., aged 44 years, married, a manufacturer.
*History*

*Complaints*—tendency to put on weight easily.
*Family history*—both father and mother are hypertensive and obese.

*Progress*

| *Date* | *Blood pressure (mm. Hg)* | *Pulse rate* | *Wt. (lb.)* | *Treatment (daily)* |
|---|---|---|---|---|
| 1959 | | | | |
| 18 Dec. .. | 138/102 | 84 | 167 | Thyroid, gr. 2 |
| 1960 | | | | |
| 4 Jan. .. | 120/90 | 82 | — | Placebo |
| 19 Jan. .. | 140/108 | 96 | — | Thyroid, gr. 2 |
| 2 Feb. .. | 126/88 | 84 | — | Placebo |
| 15 Feb. .. | 130/90 | 76 | 172 | Thyroid, gr. 2 |

*Note:* The preparation of thyroid used in these experiments was that of the US Pharmacopeia, which requires an iodine content of approximately 0·2%.

COMMENT

Although the number of cases is small and although objections to this trial may be raised on other grounds, nevertheless, I regard the results as significant. I do so, not only because they are in accord with my earlier experiences in the treatment of hypertension with thyroid, but also for these additional reasons:

1. A number of clinicians in the past have reported a fall in blood pressure on thyroid medication. They include Oliver,[7] Clifford Albutt,[8] and Molnar.[9]

2. Means [10] observed a fall in blood pressure when most of the patients with myxoedema who had high initial pressures were treated with thyroid.

3. Joll[11] noted that 'following control of the thyrotoxic state, the blood pressures may rise rather than fall. Hill found that 50% of his follow-up patients had a systolic pressure of 150 mm.Hg or more and 20% had a pressure of 170 mm.Hg or more'.

4. Fatayeva[12] drew attention to a marked deficiency in thyroid function in 65% of patients with established hypertension.

5. Strisower *et al.*,[13] in their study of the physiological effects of *l*-triiodothyronine, recorded a fall in the blood pressure in all the 18 patients investigated.

6. Barnes[14] noted signs and symptoms of hypothyroidism in patients with hypertension and found that 'thyroid therapy is highly efficacious if the disease is not too far advanced'.

7. Finally, I consider the results significant because recent animal experiments support the hypothesis, first advanced in 1953, that thyroid insufficiency in relation to noradrenaline excess is the basic factor in the causation of essential hypertension.[15-17] A brief account of these experiments follows.

THYROID INSUFFICIENCY AND NORADRENALINE

In 1945 Marine and Baumann[18] showed that thiouracil depression of the thyroid of rats was followed by hyperplasia of, and increased secretion from, the adrenal medulla. At that time it was not known that there were 2 kinds of cells in the medulla, and it was assumed that the increased secretion was adrenaline. Recently, Hopsu[19] repeated the experiment in the mouse and showed that depression of the thyroid is followed by hyperplasia of the noradrenaline-secreting cells only (Fig. 4). Thyroidectomy produced the same results. Conversely, thyroid feeding caused a diminution in the volume of the noradrenaline-producing cells and diminished secretion from them (Fig. 5). Presumably this would also apply to the noradrenaline-secreting postganglionic nerve cells of the autonomic nervous system, since these are developmentally identical. Earlier, Eränkö[20] had shown that thyroid feeding caused a selective depletion of noradrenaline from the medulla in much the same way, though not as rapidly, as insulin causes a selective depletion of adrenaline.

That a similar adrenomedullary-thyroid relationship exists in man is suggested by the reactive enlargement of the thyroid gland which may occur during noradrenaline infusions, and at the height of the paroxysm of hypertension in phaeochromocytoma.[16]

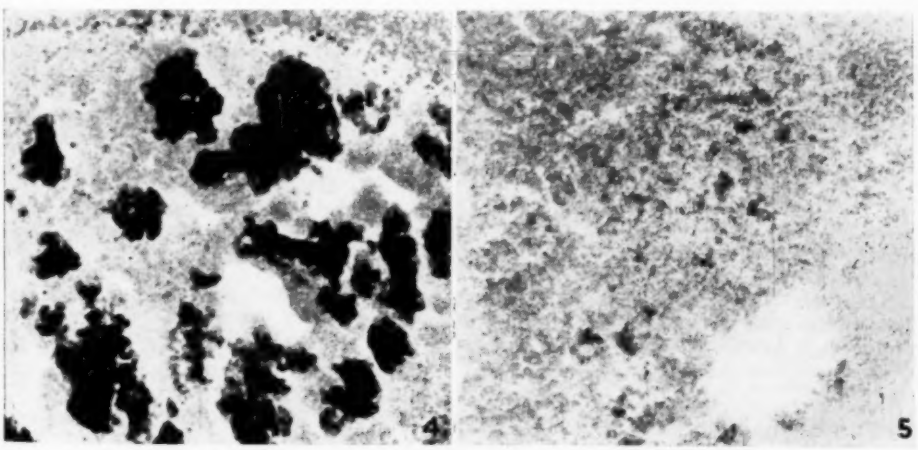


*Fig. 4.* Adrenal medulla of a mouse treated with thiouracil for 4-5 months — iodate reaction. Amount of iodate-positive tissue (= noradrenaline-secreting) increased and intensity of iodate reaction increased (after Hopsu).
*Fig. 5.* Adrenal medulla of mouse treated with thyroid powder for 3 months. Iodate-positive tissue (= noradrenaline-secreting) very scanty. The whole medulla is colourless (after Hopsu).

### DIAGNOSIS OF THYROID INSUFFICIENCY

It should be obvious from the facts just considered that thyroid insufficiency in the early stages of essential hypertension must be relative rather than absolute. Thyroid-function tests, therefore, are unlikely to prove helpful in diagnosis. How, then, are we to recognize the condition in its earliest phases? I suggest that the following indications might assist us:

1. A family history of hypertension or of any of its cerebral, cardiac or renal complications; a family history of obesity.

2. A tendency in the young or middle-aged to put on weight easily.

3. Complaints of undue fatiguability and substernal discomfort. (The classical symptoms of essential hypertension, namely, headache, giddiness, insomnia and palpitation are unlikely to be present, but their appearance, alone or in combination, particularly if precipitated by emotional stress, should be regarded with suspicion.)

4. The following objective findings: obesity, a diastolic pressure of 90 mm.Hg or over, and possibly an elevated serum-cholesterol level (above 200 mg. per 100 ml.).

With the help of these indications it should be possible to diagnose most of the cases sufficiently early to prevent the more serious consequences of this disorder.

### THE RÔLE OF NORADRENALINE IN THE CAUSATION OF ESSENTIAL HYPERTENSION

That fears and anxieties may precipitate essential hypertension and may also suddenly raise the blood pressure to high levels in established cases, has been frequently reported by practising physicians and has been confirmed in recent years by the contributions of the psychosomatic school. Such rapid elevation of the blood pressure can occur only through the intermediation of the sympathetic nervous system. Since one of the transmitters of this system is noradrenaline, there can be little doubt that the elevation of the blood pressure here is from noradrenaline excess or an excessive reaction to noradrenaline.

This conclusion narrows the aetiological gap between phaeochromocytomic and essential hypertension. Clinically, the 2 conditions are indistinguishable — a point remarked upon by a number of observers.[21,22] In a recent case of mine, where death followed the removal of a phaeochromocytoma from the left adrenal, the postmortem findings were identical with those of long-standing essential hypertension. During the 15 years before his death this patient had been diagnosed repeatedly as having essential hypertension and had survived a cerebrovascular accident (hemiplegia with residual hemiparesis) and a myocardial infarction. If, then, the conditions cannot be distinguished in life and are inseparable in death, it follows that they are caused by fundamentally similar, if not identical, mechanisms.

It must be admitted, however, that they do differ in one respect, and that is in the amount of noradrenaline found in the urine. In most cases of phaeochromocytoma this is grossly in excess of normal, while in essential hypertension a slight elevation or none at all is the rule. In recent years even this distinction has ceased to be valid, because proved cases of noradrenaline-secreting phaeochromocytomas have been described, where at no stage in the development of the disorder has it been possible to demonstrate an increased excretion of this catechol amine.[23,24]

This suggests that the difference between the majority of the cases in these 2 disorders is not of great significance. It might well admit of some simple explanation, such as an abnormality in the rate or the method of inactivation of noradrenaline.

In this connection it is of interest to recall that Goldzieher[25] described the presence of phaeochromocytomas in chronic hypertension in his book *The Endocrine Glands:* 'The adrenals in hypertension are usually enlarged and heavier, but definite hyperplasia of the medulla is seen even in glands of average or less than average size. Histological evidence of hyperplasia repeatedly offered by the writer has recently been confirmed by Frei, Lacadou and others. The proliferation of both the medulla and extra-adrenal phaeochrome tissue is either diffuse, or produces small discrete nodules only of microscopic size. These nodules differ only in size from those found in paroxysmal hypertension . . .'.

This pathological evidence would suggest that phaeochromocytomic hypertension is merely an exaggerated form of essential hypertension.

### SUMMARY

1. Further evidence is led in support of the hypothesis that thyroid insufficiency in relation to noradrenaline excess is the basic factor in the causation of essential hypertension.

2. The reason for the difficulty experienced in obtaining decisive results in the treatment of hypertension with thyroid is indicated.

3. The above clinical trial indicates that this difficulty may be overcome if essential hypertension is recognized early and treated before renal hypertension supervenes.

### OPSOMMING

1. Verdere bewyse word aangevoer ter stawing van die hipotese dat tiroïed-tekort, in aansluiting aan 'n oormaat van noradrenalien, die grondliggende oorsaaklike faktor is by essensiële hipertensie.

2. Die rede vir die moeilikheid wat ondervind word om beslissende resultate te verkry by die behandeling van hipertensie met tiroïed, word aangedui.

3. Die voorgaande kliniese ondersoek dui aan dat die moeilikheid oorkom kan word as essensiële hipertensie vroeg raakgesien word en as behandeling toegepas kan word voordat renale hipertensie intree.

I should like to thank the Warner-Lambert Pharmaceutical Company, Morris Plains, New Jersey, USA, for the supply of dummy tablets of their thyroid preparation, and the Photographic Unit of the Department of Medicine of the University of the Witwatersrand for their assistance with the illustrations.

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# THE TREATMENT OF CONGENITAL HYDROCEPHALUS

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This paper deals with our experience in the treatment of congenital hydrocephalus by means of the ventriculo-auricular-shunt operation, which was introduced into this country about 3 or 4 years ago.

Hitherto, the treatment of congenital hydrocephalus has given uniformly and universally poor results. No fewer than 30 different methods of surgical approach to the problem are known and none enjoyed popularity for any length of time. In fact it has taunted the medical profession since the days of Hippocrates. The multiplicity of methods of approach to this problem is an index of how difficult this neurosurgical problem actually is.

## ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

Experimentally, it has been shown that the cerebrospinal fluid (CSF) is not a simple filtrate, but is actively secreted by the choroid plexus. The absorption of the CSF, on the other hand, is a combination of filtration and osmosis. CSF is continually secreted by the choroid plexus in the ventricles and circulates from the lateral ventricles through the foramen of Monro into the third ventricle. From there it passes through the aqueduct of Sylvius into the fourth ventricle, from where it escapes through the medially placed foramen of Magendie and the laterally placed foramina of Luschka into the subarachnoid space. About one-fifth of the total quantity

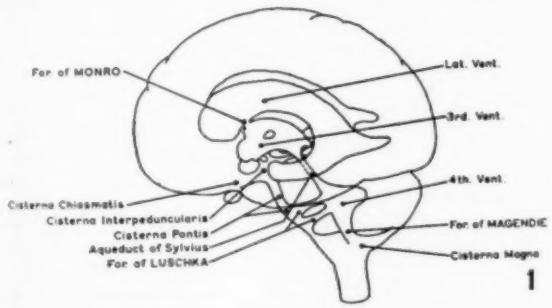



*Fig. 1.* Diagram of the ventricular system of the brain.

circulates over the spinal cord and is absorbed by the perineural lymphatics. The major portion finds its way into the basal cisterns, from where it is 'pumped' over the surface of the brain and absorbed by the arachnoid villi. Fig. 1 is a diagrammatic representation of the ventricular system in the brain.

## PATHOLOGY

Pathologically, we recognize 2 types of hydrocephalus:

1. *Compensatory Type*

In this type there is usually no enlargement of the head and no increase in the CSF pressure. In the newborn it is caused by agenesis of the brain, and its counterpart is found in the adult as cerebral atrophy. There is passive distention of the ventricular system and laking in the subarachnoid space. It is of very little importance clinically to us and is not amenable to treatment.

2. *Hypertensive Hydrocephalus*

This is caused by: (*a*) excessive formation of CSF, (*b*) obstruction to its circulation, or (*c*) defective absorption.

According to Russell, the majority of cases in this group are due to some form of obstruction. Here, there are 2 sub-groups:

A. *The communicating type of hydrocephalus.* This is readily recognized clinically by the amount of CSF obtained by lumbar puncture. If more than 3 - 4 ml. are obtainable, then this is a communicating type of hydrocephalus. When neutral phenolsulphophthalein is injected into the ventricles, it will appear in the lumbar CSF in 3 - 5 minutes. The pneumo-encephalogram in this type of case shows that the ventricular system fills readily, and if the subarachnoid space is filled, the level of the obstruction can sometimes be easily demonstrated. The obstruction is usually at the incisura tentorii, the interpeduncular or chiasmatic cisterns.

B. *The non-communicating type of hydrocephalus.* This is practically always caused by some obstruction of the foramen of Magendie or stenosis of the aqueduct of Sylvius. Usually less than 3 - 4 ml. of CSF is obtained at lumbar puncture and neutral phenolsulphophthalein injected into the ventricles will not readily appear in the lumbar CSF. A lumbar pneumo-encephalogram will either fill the fourth ventricle only, in the case of aqueduct stenosis, or will not fill the ventricular system at all, in the case of obstruction at the foramen of Magendie.

## CLINICAL PICTURE

The diagnosis of hydrocephalus is made without difficulty because of the obvious enlargement of the head and bulging of the anterior fontanelle. The enlargement is confirmed by measuring the occipito-bregmatic circumference, which will be above the normal average. As the hydrocephalus becomes more marked, the disproportion between the normal face and large cranium becomes more striking. The fontanelle and

sutures widen, the scalp veins become distended, the eyes are displaced downwards from pressure on the orbital plate, and the sclera becomes visible above the iris. The patient becomes irritable and has a high-pitched cry. Feeding becomes a problem, and there is relative weight loss of the body as the head grows in size, even up to a point where there is frank emaciation and dehydration.

Optic atrophy and mental retardation are in inverse proportion to the rapidity of the production of the hydrocephalus, but we are all familiar with the mild hydrocephalic with an I.Q. above the average.

Some hydrocephalic enlargements become spontaneously arrested weeks or months after onset. Every case should be assessed on its own merits to decide when to operate. It is our practice to observe the baby for at least 2 weeks, when circumstances are favourable, and if there is a steady and regular increase in the size of the head it is unlikely that spontaneous arrest will ensue.

All the patients must be subjected to investigations, e.g. X-rays of the skull, subdural taps, and pneumo-encephalography.

## SURGICAL APPROACH

Fifty years ago, Payer attempted a ventricular-venous anastomosis. All 3 of his patients died within 4 months.

Six years ago, Pudenz *et al.* developed a ventricular-venous shunt by introducing one end of a catheter into the lateral ventricle and the other end into the right auricle of the heart (Fig. 2). The distal tip, which is lodged in the auricle (Fig. 3), has a slipcore-valve mechanism which will allow unidirectional flow of CSF. Since this method is an imitation of nature to shunt the CSF into the venous system, it is more likely to meet with success than other methods whereby the CSF is shunted into one or other body cavity, e.g. Eustachian tube, extradural space, pleural cavity, peritoneal cavity, lesser sac, ureter, duodenum or colon.

*Fig. 2.* Diagram showing the ventricular-venous shunt *in situ*.

The method described by Pudenz involves joining the appropriate length of the proximal and distal ends of the catheter by means of a metal connection in the neck.

### *Causes of Failure*

Four of our first 8 patients had to be re-explored at various times, but since we introduced a modification of the Pudenz method, whereby the junction of the proximal and distal ends of the shunt is made immediately below the burr hole or eliminated altogether, the recurrence rate has dropped to less than 25% in 20 successive cases.

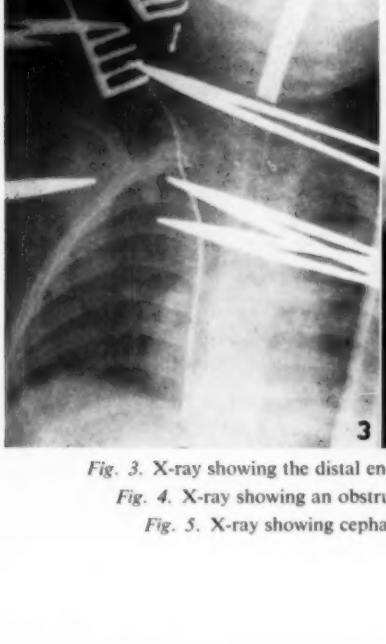
In 4 of the patients needing re-exploration we found:

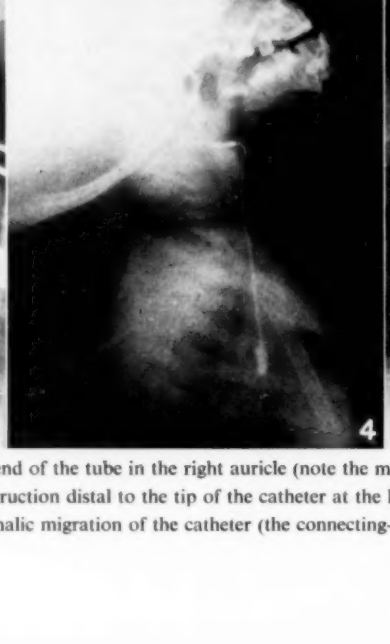
(*a*) a kink in the tubing at the metal junction (associated in 2 cases with a crack in the tube at the bend so produced);

(*b*) blocking of the lumen of the tubing by brain tissue;

(*c*) blocking or sealing off of the valve by fibrous tissue from the wall of the superior vena cava caused by faulty introduction—the tip of the shunt was not introduced into the right auricle, but was still in the superior vena cava (Fig. 4). The tube can also migrate upwards towards the head from the auricle (Fig. 5).

In a few cases the shunt becomes blocked by particulate matter. A flushing device has recently been introduced which already shows marked improvement in the ultimate results. It consists of a flanged silicone capsule and a diaphragm valve, which is introduced into the shunt where it comes through the burr hole immediately under the skin.



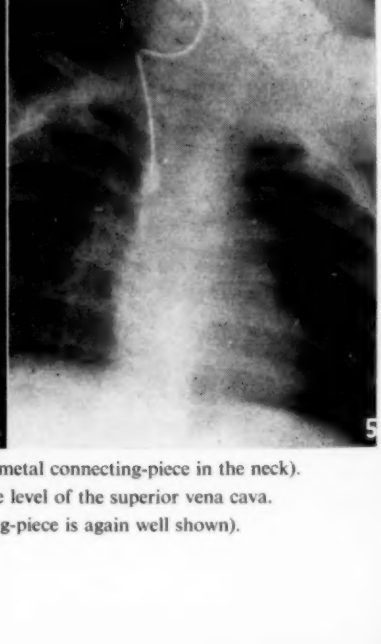




*Fig. 3.* X-ray showing the distal end of the tube in the right auricle (note the metal connecting-piece in the neck).

*Fig. 4.* X-ray showing an obstruction distal to the tip of the catheter at the level of the superior vena cava.

*Fig. 5.* X-ray showing cephalic migration of the catheter (the connecting-piece is again well shown).

When the capsule is compressed by pressure exerted on the overlying skin, the diaphragm closes the ventricular inlet and the CSF in the capsule is forced through the cardiac tube.

The operation, as such, is technically easy to perform and hardly disturbs the baby. No baby requires transfusion, and it should be avoided for fear of over auto-transfusion from a large reservoir in the head.

*Results*

We had 1 postoperative death in 30 cases and 36 operations. At postmortem examination, a massive intraventricular haematoma was found, associated with anaemia and collapse of the cerebrum. The greatest danger is intercurrent infection and septicaemia.

SUMMARY

The horrifying condition of congenital hydrocephalus can now be successfully treated in many cases, and the foetus with the large head need not be destroyed by the obstetrician, nor need the hydrocephalic patient, discovered after birth, be left to die a slow death.

Our experience in 30 cases over a period of 3 years is most encouraging, and should the shunt need to be replaced or adjusted as the child grows, this should offer no difficulty to either surgeon or patient.

# SERUM TRANSAMINASE LEVELS IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION

H. E. A. MENTZ, D.Sc. (PRET.), A.R.I.C., *South African Institute for Medical Research*, and A. L. AGRANAT, M.D. (DUBL.), F.R.C.P. (EDIN.), D.T.M. & HY. (RAND), *Johannesburg*

Glutamic oxalacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) are widely distributed in the human body.[1] In comparison with the levels in the tissues, the normal serum levels of both enzymes (SGOT and SGPT) are very low. They are liberated into the blood in increased quantities on death of the cells in which they occur, and increased serum levels have been found following necrotic lesions in many different organs. Estimations of SGOT have been used most often in investigations of myocardial infarction and SGPT in liver-cell necrosis; the GPT content is greatest in liver tissue and less in heart muscle, and the increase in SGPT is thought by some to exceed that of SGOT in hepatic disorders.[2]

It has been reported that a raised SGOT level may confirm the diagnosis of myocardial infarction.[3-5] The level reaches a peak which may be 15 times higher than normal within 24 - 48 hours, and returns to normal by the 4th - 7th day.

It has been suggested that with relatively small infarcts the peak level may be within or at the upper limit of the accepted normal range, especially if a patient's serum transaminase in health is in the low range of normal; for this reason serial determinations should be carried out.

Sometimes it is difficult to distinguish on the electrocardiogram (ECG) between coronary insufficiency (ischaemia) and limited infarction. The SGOT level should be of diagnostic help in such cases, since it was found to remain unaltered if ischaemia is not accompanied by necrosis.[5] Dewar *et al.*[7] found that the enzyme estimation is complementary to, but not necessarily more reliable than, the ECG in showing whether necrosis has taken place.

The SGOT level may also be of special value in complicated infarction. A second infarction will give a secondary peak, even though the ECG does not change.[3,6,7] Also, in some patients with previous infarction, the ECG can be so distorted that on a single initial tracing alone it may be impossible to deduce with certainty that fresh infarction has occurred. According to Keele *et al.*[8] anginal pain at rest with an abnormal, but not diagnostic, ECG associated with a raised SGOT concentration, carries the same prognostic significance as does the electrocardiographically proved infarction, while the transaminase level does not tend to be raised with chest pain from most other causes.

In this investigation we attempted to answer the following questions:

1. When is the SGOT estimation of help in the diagnosis of myocardial infarction?
2. What are the minimum number of tests required in a single case?
3. What additional information can the SGPT estimation supply?

The answers to the questions are important. If they are known, the test need not be done unnecessarily, the number of tests may be reduced to a minimum, and the interpretation of the test may be properly assessed.

*Method of Investigation*

Two groups of patients were investigated on a fairly uniform basis. Apart from 6 patients treated in the Johannesburg General Hospital, all the patients were admitted to a nursing home where they were under the personal care of one of us (A.L.A.). Electrocardiograms were done and repeated where necessary. The SGOT and SGPT levels were estimated daily for 10 consecutive days and the values were correlated with the clinical findings. In order to reduce the transaminase estimations to a practical number, the SGOT and SGPT levels were assessed in most cases for the first 4 days in each of the series. These values represent 4 daily consecutive levels in blood collected on admission, and as soon after the episode of chest pain as possible. To prolong this investigation to 6 - 10 consecutive days makes the test, in our opinion, impracticable in most instances. In fact, if a single diagnostic level should be positive, no more estimations need be done.

*Methods Used for Transaminase Estimations and Normal Values*

SGOT and SGPT levels were estimated according to the method of King.[10]

In a series of 36 normal adult males and females the SGOT level was between 18 and 100 units, and the SGPT level between 18 and 89 units. This is in accordance with the normal figures reported by King.

RESULTS

*Group A*

In this group, which consisted of 20 patients, the diagnosis of myocardial infarction could be made clinically and on ECG evidence. Daily transaminase tests were carried out to assess their value.

In 16 of the 20 patients, positive ECG evidence and positive transaminase levels were obtained. In 4 patients the SGOT values failed to reach diagnostic levels. Significantly, these 4 were patients with subendocardial necrosis, as demonstrated by the electrocardiograms. Presumably the liberation of GOT into the blood stream may be related to quantitative necrosis of heart muscle. The 4 cases are briefly quoted:

1. *S.L.F., female, aged 50 years* — admitted with a typical attack of myocardial infarction. The ECG showed a posterior infarction. The SGOT levels for the first 3 consecutive days were 50, 50, and 40 units per 100 ml.
2. *J.H.S., male, aged 52 years* — admitted with a severe attack of substernal pain. The ECG showed a subendocardial infarction. For the first 3 consecutive days the SGOT levels were 29, 90 and 55 units. A steep rise or fall in the SGOT levels in the region below the diagnostic level is considered significant by some authors; however, it is apparent that the ECG evidence was more important than the SGOT levels in this case.
3. *H.T., female, aged 52 years* — the description of pain on admission was suggestive of myocardial infarction. The electrocardiograms showed a subendocardial infarction. The SGOT levels for the first few days ranged from 50 to 75 units per 100 ml.
4. *O.R.S., male, aged 55 years* — chest pain was typical of myocardial infarction. Serial electrocardiograms showed a subendocardial infarction. The SGOT levels ranged from 40 to 70 units per 100 ml.

These results show that the transaminase test may be unnecessary if the diagnosis of myocardial infarction can be confirmed by a positive ECG. It is also significant to note that in 4 cases of subendocardial infarction the SGOT concentrations did not reach diagnostic levels. In case 2, a steep gradient occurred below the accepted diagnostic level — this may have indicated a small infarction.

On the whole, the SGPT levels obtained were lower than the corresponding SGOT levels.

*Group B*

This group was made up of 20 patients with suspected or recurrent myocardial infarction.

The transaminase test is important in these cases. As already mentioned, no further tests are as a rule necessary if the ECG is unequivocally positive for myocardial infarction. However, where atypical pain occurs without confirmatory clinical or ECG evidence, these tests have their greatest value. For practical purposes the test need not be applied to this group in general; it is only when doubt exists that the transaminase tests are needed.

The following are examples of some situations where the test was of help:

*Case 1. Recurrent myocardial infarction.* Male, aged 42 years — admitted with paroxysmal fibrillation and a history of previous myocardial infarction. The patient had chest discomfort, but no severe pain. The SGOT level was 140 units per 100 ml. Recurrent infarction was diagnosed, which was later supported by a changing pattern in the ECG.

*Case 2. Attacks of paroxysmal fibrillation and substernal pain for 10 minutes in a patient with a previous myocardial infarction.* Male, aged 51 years — he was distressed, with a feeling of tightness in the chest, and dyspnoea. The SGOT levels ranged from 65 to 70 units. Electrocardiograms showed a normal rhythm and an old myocardial-infarction pattern. There was probably no recurrence of infarction.

*Case 6. Recurrent chest pain with bundle branch block from preceding myocardial infarction.* Male, aged 55 years — there was previous myocardial infarction with residual left bundle-branch block, and the electrocardiograms were unchanged after recurrent bouts of chest pain. The SGOT level was 160 units and a diagnosis of recurrent infarction was made.

*Case 7. Recurrent 'attack' from 'food poisoning' resembling previous myocardial infarction.* Male, aged 55 years — he had a previous myocardial infarction and the ECG also showed an old myocardial-infarction pattern. He had recurrence of severe upper abdominal and lower substernal pain with sweating, vomiting and diarrhoea, and was shocked. The blood pressure fell from 190/110 to 110/90 mm.Hg. The patient ascribed the symptoms to 'food poisoning', but clinically the diagnosis was strongly in favour of a recurrent infarction. Three consecutive SGOT tests showed levels of 50, 70 and 40 units, which meant that the patient's diagnosis was correct.

*Case 13. Previous angina pectoris, admitted with severe substernal pain.* Female, aged 50 years — the patient was a very anxious type of person. The ECG was normal and the SGOT levels ranged from 50 to 70 units. Myocardial infarction was excluded.

*Case 16. Short period of severe substernal pain followed by a 'blackout' in a patient with recent myocardial infarction.* Male, aged 34 years — shortly after an attack of myocardial infarction the patient was admitted with the above symptoms. Clinically there was no shock. The ECG showed an extensive anterior myocardial-infarction pattern similar to that of the previous record. He recovered rapidly. The SGOT values ranged from 45 to 83 units, which excluded recurrence of myocardial infarction.

The following are examples of some situations where the test was of no help:

*Case 4. Where the test is done more than 3 days after an acute episode.* Male, aged 55 years — he had myocardial infarction 3 years previously, and had received long-term anticoagulant therapy since then. He had a sudden recent 'collapse' with diarrhoea and vomiting and was unconscious, but did not have pain. His blood pressure fell from 160/105 to 105/60 mm.Hg. Serial electrocardiograms showed an old myocardial-infarction pattern. The SGOT level, which could only be done 5 days later, was 85 units. Clinically the diagnosis was assessed as re-infarction. The SGOT test had probably lost its value on account of the 5-days' time lag.

*Case 11. Where the ECG evidence takes preference to the transaminase test on absence of typical clinical features of myocardial infarction.* Male, aged 49 years — he had typical anginal pain and there were slight ischaemic changes on the ECG at rest. Exercise produced a typical pattern of acute posterior myocardial infarction, but without prolonged pain or shock. The daily sequence of the SGOT levels for 9 days was 95, 20, 20 - 65 - 50 units. The SGPT levels were all lower. The SGOT concentration did not reach a diagnostic level of more than 100 units, although the significance of the steep fall from 95 to 20 could not be discarded. Subsequent electrocardiograms confirmed a posterior myocardial infarction. In this case the ECG showed myocardial infarction before any conclusions could be drawn from the transaminase tests.

*Where extraneous pathology nullifies the value of the transaminase test — cases 14, 15 and 17.*

*Case 14.* Male, aged 34 years — he had severe substernal pain a week before admission. The ECG was normal at rest, but showed significant ST depression after exercise. Chest pain occurred during the last week of a six-week period of bed rest for infectious hepatitis. For 10 days the SGOT values ranged from 290 to 440 units. The SGPT values ranged from 460 to 500 units. Liver-function tests showed gross hepatocellular damage.

Subsequent electrocardiograms confirmed the diagnosis of coronary ischaemia and not that of myocardial infarction. In such a case, therefore, the transaminase test can be of no help.

*Case 15.* Female, aged 61 years — she had severe substernal pain on admission, with a history of several preceding attacks of myocardial infarction. She presented with a clinical picture of myocardial infarction, but the ECG was normal. The SGOT level on admission was 500 units, which was difficult to fit in with a normal ECG.

For the next 6 days the SGOT levels ranged from 190 to 500 units, while the SGPT curve was even higher and more sustained, with levels ranging from 320 to 470 units.

Four subsequent electrocardiograms during convalescence were normal, except for a mild ischaemic pattern on exercise. When we heard that the patient had had a previous cholecystogram, we performed a cholangiogram which showed an obstructive process at the ampulla of Vater with dilatation of the common duct. Liver-function tests and serum-amylase levels were normal. A barium meal and a X-ray of the chest did not show any abnormalities. As the case developed a diagnosis of myocardial infarction could not be sustained. This most likely also applied to the preceding occasions when myocardial infarction was diagnosed. The extraneous pathology here was an acute cholangitis with obstruction of the common bile duct, resembling myocardial infarction symptomatically. The SGOT levels may have been misleading at the onset, but this impression was soon corrected. A point of note here is that the SGOT values seldom reach these very high levels in myocardial infarction.

*Case 17.* Male, aged 52 years — on the second day after a cholecystectomy the patient complained of anterior chest pain and then 'collapsed'. He was pulseless, dyspnoeic, and cyanosed, while the chest was full of bubbling râles. Intravenous 'levophed' was then given. On the following day the electrocardiogram suggested a myocardial-infarction pattern (biphasic T waves but no ST displacement) in $V_3$ and $V_4$. Clinically, a postoperative myocardial or pulmonary infarction was suspected, but the ECG showed no evidence of acute cor pulmonale. An X-ray of the chest showed pulmonary infarction. A second ECG was again compatible with a small anterior myocardial infarct. The SGOT levels during the first 3 days ranged from 150 to 195 units, while the SGPT levels ranged from 160 to 290 units. After a week the transaminase tests were repeated; for 4 days the SGOT levels ranged from 125 to 150 units and the SGPT levels from 205 to 270 units. An extracardiac cause is usually suggested when the SGPT curve is higher than the SGOT curve, and the sustained high levels of both enzymes also support an extracardiac cause. A third ECG was normal.

The 'collapse' and ECG evidence of myocardial infarction suggested that this was the predominant pathology. The extraneous pathology (pulmonary infarction with bronchopneumonia) nullified the value of the SGOT test.

The SGPT levels were invariably lower than the SGOT levels in those cases where no mention has been made of the SGPT values.

## DISCUSSION

From a study of these cases certain points emerge which are of importance in assessing the value of the test.

When the clinical findings and ECG are diagnostic of myocardial infarction, there is no necessity to do the transaminase test. The electrocardiographic evidence is in general far more important than the transaminase tests. These may only confirm the diagnosis where it is already established.

In Group A the transaminase tests were done to study their significance in known cases of myocardial infarction. Another 20 patients were studied in Group B, which included the 'problem cases' where a straightforward ECG in most cases could not prove the diagnosis of myocardial infarction without discussion.

The SGOT level exceeded the diagnostic level of 100 units within the first 3 days in most cases of myocardial infarction. It did not reach a diagnostic level in 4 cases of subendocardial infarction which were proved by electrocardiograms. A steep gradient in the subdiagnostic zone may be significant, but this has not been definitely proved.

The SGOT estimations were usually of little value after the first 3 days following a myocardial infarct. A later rise, without recurrence of chest pain, could usually be ascribed to some extracardiac cause.

For the diagnosis of myocardial infarction, the SGPT estimations were of no additional value.

When a raised SGOT level is accompanied by an even higher level of SGPT, it is usually, but not always, a sign of an extracardiac cause. Very high SGOT levels, e.g. 400 - 500 units, were seldom found in cases of myocardial infarction. For differential diagnostic purposes both tests should be done. When the levels for both SGOT and SGPT remain sustained for 7 days or more, the tests usually indicate an extracardiac cause, with or without an associated myocardial infarct.

When an electrocardiogram is unobtainable, 3 consecutive daily SGOT tests should be of considerable value in confirming a clinical diagnosis. This applies in particular to doctors practising in an area where an ECG is not available.

## SUMMARY

A clinical trial was undertaken to assess the indications, value, and limitations of the serum-transaminase tests in the diagnosis of myocardial infarction. It was concluded that:

1. The transaminase tests are unnecessary if the ECG is diagnostic of myocardial infarction.
2. In general, the tests are required in only a limited number of cases, usually only in those cases where the ECG is equivocal.
3. The SGOT values may not reach diagnostic levels in cases of subendocardial infarction.
4. The SGOT value usually reaches a diagnostic level within the first 3 - 4 days of an episode of myocardial infarction. A secondary rise without recurrence of chest pain is probably no proof of recurrent myocardial infarction.
5. Very high SGOT levels, e.g. 400 - 500 units, are seldom found in pure myocardial infarction. These usually indicate extracardiac pathology, especially when accompanied by even higher SGPT levels, and when both levels are sustained for a period of 7 days or more.
6. The SGPT estimations were of no additional help in the diagnosis of myocardial infarction.
7. To confirm a diagnosis of myocardial infarction, serial SGOT estimations should be of considerable value

to doctors practising in an area where an ECG is not available.

We wish to thank the Director of the South African Institute for Medical Research for his interest in this investigation.

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# SPONTANEOUS EXTERNAL BILIARY FISTULA

S. MOVSAS, M.B., B.CH. (RAND), F.R.C.S. (ENG.), *Department of Surgery, University of Natal, Durban*

External biliary fistulae may arise as a complication of gallbladder surgery, may be purposefully established, or may, very rarely, be spontaneous.

The first case report of a biliary fistula following perforation of gallstones through the abdominal wall is accredited to Thilesus in 1670 (quoted by Henry and Orr[5]). By 1890 Courvoisier had published a series of 499 cases of perforation of the gallbladder, and of these 169 were listed as spontaneous external abdominal fistulae. Henry and Orr[6] reported a case of spontaneous biliary fistula and collected a further 36 cases from the world literature between 1890 and 1948. From 1948 to 1960 inclusive only 3 additional cases[2,6,8] could be found in the world literature.

Because of the increasing rarity of the condition the following case is reported and the literature is briefly reviewed.

## CASE REPORT

The patient, an Indian male aged 28, was admitted to hospital on 8 April 1960, complaining of colicky upper abdominal pain and vomiting. During a previous admission he had been operated on for intestinal obstruction from worms. On examination he was pyrexial (101°F.) and the right hypochondrium was tender and rigid. Rebound tenderness could be elicited in this area. There was no evidence of jaundice. The haemoglobin was 15·7 G. per 100 ml., the leucocyte count 22,000 per c.mm., and the serum bilirubin 1·1 mg. per 100 ml.

A definite diagnosis was not made and laparotomy was decided on. Through a right upper paramedian incision, adhesions of omentum to the anterior abdominal wall were dissected free and revealed an acutely inflamed, but not unduly distended, gallbladder. This was palpated, but gallstones were not detected within it or within the common bile duct. The gallbladder was not drained or removed, since the surgeon performing the operation decided that the degree of distension did not appear to warrant a drainage procedure. Instruments were not applied to the gallbladder, and the omentum surrounding it was not stripped away. The rest of the laparotomy was normal and there was no evidence of pancreatitis. The abdomen was closed in layers without drainage. Chloramphenicol, 100 mg., was added to each 1,000 ml. of intravenous infusion.

On the fourth postoperative day the patient had a high pyrexia, with persistent tenderness and guarding in the right hypochondrium. The next day he experienced a sudden bout of colicky pain, which was followed a day later by a discharge of bile-stained fluid from the lower end of the operation wound. By the 12th day the temperature had become normal and by the 26th day there was hardly any discharge from the wound, and he was sent out of hospital.

On 14 May 1960 the patient complained of a marked purulent discharge from the wound and was re-admitted. On examination a sinus discharging pus (but not bile) was seen in the centre of a healed right upper paramedian incision. A specimen taken from the sinus was cultured and showed a heavy growth of *Staph. pyogenes* and *B. coli*. The haemoglobin was 13·9 G. per 100 ml., and the leucocyte count 10,000 per c.mm. On 6 June a cholecystogram was performed using 'biligrafin'. This showed that the hepatic and common bile ducts could be fairly adequately visualized, but the gallbladder was not seen at any stage of the examination, which was continued for almost 3 hours. On 1 July it was decided to explore the sinus, which was discharging mucoid material. After its wall of granulation tissue had been scraped away a catheter was inserted into the sinus. Through the catheter lipiodol was injected and appeared to pool in a small cavity deep to the abdominal wall. The patient was discharged from hospital on 8 July.

On 10 November (7 months after the first admission) the patient was re-admitted, still discharging pus from the sinus, and on 18 November the sinus was explored and was found to extend into the gallbladder, which contained a solitary stone. A cholecystectomy was performed and the biliary

*Fig. 1.* Photograph of operative specimen showing a length of string in the biliary fistula, the chronically inflamed gallbladder to the left and the skin of the anterior abdominal wall to the right. On the extreme right is the gallstone cut in half. The rule shown is graded in cm.

fistula was excised at the same time (Fig. 1). On palpation of the common bile duct, gallstones were not detected. When the patient was discharged from hospital on 2 December, an area of superficial skin death was healing satisfactorily. Later (7 January 1961), when the patient was seen in the follow-up clinic, the abdominal wound had healed completely.

## DISCUSSION

Spontaneous external biliary fistulae occur more frequently in women than in men, and are found most commonly in persons in the 5th - 7th decades. Most commonly the pathogenesis is an acute suppurative cholecystitis or empyema of a gallbladder containing gallstones. The fundus, which becomes adherent to the anterior abdominal wall, ruptures, discharging pus and gallstones which burrow through the abdominal wall to become discharged later; thus establishing external fistulae at varying sites. The

usual site of the external opening is the right hypochondrium. Less commonly the fistulous opening has been reported in the region of the umbilicus,[4] the right iliac fossa,[2] the right thigh,[1] and the left lumbar region.[7] Associated gallstones were present in all the cases collected by Henry and Orr.[5] The discharge from the fistula may contain only mucus (where the cystic duct has been blocked), or bile, pus, or one or more gallstones.

Confirmation of the diagnosis is best made by injecting lipiodol through a catheter which has been inserted into the fistula, and by this means demonstrating a communication with the gallbladder. There were no deaths in the series collected by Henry and Orr[5] and spontaneous healing occurred in 20% of their cases. Despite this, cholecystectomy, coupled with complete excision of the fistulous tract, is the recommended treatment of choice.

It is stressed that at no time, in the case reported here, was the gallbladder interfered with, nor was the surrounding inflammatory tissue disturbed. Certainly instruments did not go near the vicinity of the gallbladder during the operation. It is interesting that in this case gallstones were not reported at the time of the first exploratory laparotomy (8 April 1960). By the time the cholecystectomy was performed 7 months later, a single gallstone had formed by precipitation of bile salts and pigments in a gallbladder with an obstructed cystic duct. It is suggested that the increasing rarity of spontaneous biliary fistulae in recent years may be attributed to increased operative intervention in acute cholecystitis.

SUMMARY

A case of spontaneous external biliary fistula is reported. A brief review of the literature is included.

My thanks are due to Prof. A. E. Kark for his advice and criticism, and to Dr. S. Disler for permission to publish this case.

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## IN MEMORIAM

HARRY MENTER, M.B., CH.B., B.A.O.

*Dr. Eugene Baskind of Johannesburg, writes:*

Dr. Harry Menter died suddenly on 10 June 1961 at the age of 45 years, from a coronary thrombosis. Harry Menter was born in Johannesburg and qualified with the degrees M.B., Ch.B., B.A.O. at Trinity College, Dublin. After qualifying he worked as a general practitioner in Doncaster for a time, and then undertook specialized work in gynaecology and obstetrics under Professor Canney at Cambridge Hospital. He returned to South Africa in 1942 and commenced practice in Johannesburg. He continued to maintain his great interest in obstetrics by working at the Bridgeman Memorial Hospital and at the Gospel Mission Hospital. In 1947 he returned to England for a year to do postgraduate work. On his return, and until his death, he carried on a busy general practice.

Dr. Menter

Harry Menter had a quiet, unassuming and friendly manner. This, together with his undoubted capabilities, kindness and gentleness towards the sick of all races, inspired confidence in patients and made him a most sought-after doctor. However, he was never much interested in the material rewards of practice; his goal was a high standard of medical attention to each and every one of his patients, and he continually maintained this ideal. His patients have indeed sustained a great loss.

The integrity, principles and high ideals which characterized his professional life were maintained in all other spheres and were reflected in his home and community life.

His interest in his family's sporting activities was well known and his presence at all events in which his children participated gave them the wonderful encouragement which has helped them to attain prominence in their school and university teams.

To his wife and family we express our deepest sympathy.

## PASSING EVENTS : IN DIE VERBYGAAN

*Audio-Digest Tape-recording Service.* A new Audio-Digest tape recording, on the 12th Mental Hospital Institute held at Salt Lake City, Utah, USA, has been received from SKF Laboratories, and is now obtainable from the Business Manager, Medical Association of South Africa, P.O. Box 643, Cape Town.

* * *

*South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting.* The next meeting will be held on Monday 2 October at 5.10 p.m. in the Institute Lecture Theatre. Dr. Basil A. Bradlow will speak on 'Observations on the rôle of red cells in coagulation'.

* * *

*Dr. T. Donald Gordon and Dr. Mungo Park Ross* will practice in partnership as ophthalmic surgeons from their new address, 1214 United Building, Cnr. Smith and Gardiner Streets, Durban, from 1 October. Telephones: Rooms 24663, 62654.

* * *

*Dr. Edward Abro,* urologist, of Johannesburg, has changed the telephone number of his consulting rooms in Medical Centre, Jeppe Street to 228686. His residential number remains 418505.

*Railway Medical Officers' Group (M.A.S.A.), Reception.* Will all Railway Medical Officers who intend attending the buffet-supper party on Tuesday afternoon 26 September at the home of Mr. Willem Steenkamp please notify Mr. Steenkamp or Dr. Alex Gordon. This information is urgently required with a view to catering.

*Spoorweggeneesheersgroep (M.V.S.A.), Onthaal.* Sal alle spoorweggeneeshere wat van plan is om die buffet aandete by die huis van dr. Willem Steenkamp by te woon, asseblief met dr. Steenkamp of dr. Alex Gordon in verbinding tree. Die informasie is dringend nodig in verband met reëlings wat getref word vir verversings.

* * *

*The Society for Endocrinology, Metabolism and Diabetes of Southern Africa* is arranging 2 symposia during the forthcoming Medical Congress. These will take place on the afternoon of Tuesday 26 September. The subjects are 'Intersex and hypercalciuria'. Following the scientific presentation, the Annual General Meeting of the Society will take place. All members and those interested are invited to attend these symposia.

*Dr. M. M. Suzman, Harveian Lecturer for 1962.* The Harveian Society of London (founded in 1831 for the advancement of medical science) arranges for the Harveian Lecture to be given once a year. The lecturer, as a rule, is selected from among the members of the Society; but occasionally a distinguished member of the medical profession outside the Society is invited to deliver the Lecture. Dr. M. M. Suzman, of Johannesburg, has been selected to give the next Harveian Lecture on Wednesday 21 March 1962. The total membership of the Society is 385. A new Commonwealth membership was recently instituted and Dr. M. M. Suzman was invited to be its first honorary member.

* * *

*Society for Clinical and Experimental Hypnosis of South Africa (Southern Section).* The next meeting of this Society will be held on Friday 29 September at 8.15 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr B. W. Levinson will speak on 'Neurophysiology of hypnosis'.

*Cecil John Adams Memorial Trust Travelling Fellowships.* The late Mr. Arthur E. Adams of Rondebosch, during his lifetime established a Trust for the endowment of Travelling Fellowships in memory of his son, Cecil John Adams, who lost his life in the last war.

A Fellowship has an annual value of about R1,800, and shall be tenable for one complete year of twelve months, but may be extended for a further period at the discretion of the Selection Committee. Four Fellowships are available.

Candidates must be South African graduates in medicine or medical science, they must have shown promise of being likely to profit from further study, research and experience in other countries, and must have been resident in South Africa for at least three years immediately prior to the date of application for a Fellowship.

Application (in triplicate) must be made on a prescribed form obtainable, and must reach the Trustee, Dr. G. F. van Tonder, 6 Church Square, Cape Town, by 31 October 1961.

## BOOK REVIEWS : BOEKBESPREKINGS

### PRÉCIS DE TOXICOLOGIE

*Précis de Toxicologie.* By René Fabre and René Truhaut. Two Volumes. 1961.Price: Vol. I, 25 NF., Vol. II, 33 NF. Société D' Edition D' Enseignement Supérieur, 5, Place de la Sorbonne, Paris 5e.

Dr. Fabre is a member of the Institute and Academy of Medicine in Paris, and Dr. Truhaut is Professor in the Faculty of Pharmacy in the same city. In their research and through their publications, both authors have materially contributed to various fields of medicine, but chiefly to toxicology. It is because of these facts that Fabre and Truhaut are eminently qualified to publish a treatise on toxicology in all its aspects.

In volume I of Précis de Toxicologie, the authors discuss the manner in which poisons penetrate into the system, their modes of action, and their distribution and localization in the system, as well as their elimination from the body.

A large number of toxic gases and vapours are dealt with, among others, phosphor and arsenic, alcohols, glycols, esters, aldehydes, halogenated hydrocarbons, hydrocyanic acid and its derivatives, phenol and its derivatives, etc.

In volume II, the authors deal with the aetiology of poisoning and with fluorides and fluosilicates, nitrates and nitrites, acids and alkalis, and combinations of arsenic, antimony, mercury, lead, copper, cadmium, thallium, chromium, manganese, zinc and barium. The toxicity, symptoms of poisoning, postmortem appearances, and methods of treatment and prophylaxis, as well as the elimination of these poisons from the body, are discussed. Valuable information concerning the methods of extraction and the solvents employed, is supplied.

This book is a valuable contribution to our knowledge of the different aspects of the above poisons and should be an asset to the library of toxicologists, medical practitioners and hospitals. D.G.S.

### NURSING

*Principles of Surgery and Surgical Nursing.* By Selwyn Taylor, D.M., M.Ch. (Oxon.), F.R.C.S. and Olga Worral, S.R.N., S.C.M., S.T.D. Pp. viii + 290. Illustrated. R1.50 net. London: The English Universities Press Ltd. Cape Town: Howard B. Timmins. 1961.

This is one of the Modern Nursing Series, the general editors of which are a well-known Professor of physiology and two prominent members of the staff of the Royal College of Nursing, London.

The series is designed to cover the requirements of the examinations of the General Nursing Council. This volume deals with the principles of surgery that a nurse should know, with particular reference to the nursing of surgical cases, in a concise yet adequate manner. The chapters are arranged systematically and the index is comprehensive.

As a book it is interesting and readable and should appeal to student nurses. A.H.T.

### SURGERY IN PORTAL HYPERTENSION

*The Surgical Treatment of Portal Hypertension, Bleeding Esophageal Varices and Ascites.* By M. Judson Mackby, M.D., D.A.B.S., F.I.C.S. Pp. xviii + 250. R8.40. Oxford: Blackwell Scientific Publications and Springfield, Illinois: Charles C. Thomas. 1960.

This monograph is one that has appeared in the American Lecture Series. It is well produced on good quality paper with excellent diagrams. It is essentially a manual of surgical therapy in portal hypertension and ascites; the indications for various operations are discussed and the technique of these operations is described in detail. The book reads easily, but suffers from two defects: There is much unnecessary repitition, and many statements are made without adequate proof. Examples of the latter are: 1. 'The suture line is all too prone to separate in any case and the operation is hazardous on this account as fatal anastomotic disruption has occurred with distressing frequency'. This is said of the gastric bi-section vagotomy and pyloroplasty operation for bleeding oesophageal varices — an operation which has many adherents outside the USA. 2. A statement that abstinence from alcohol prevents cirrhosis from progressing. 3. A statement that a side-to-side portacaval anastomosis, larger than the diameter of the portal vein, can drop the portal pressure to such an extent that thrombosis occurs at the line of anastomosis. This cannot be so because no matter how large the anastomosis, the flow cannot be greater than that allowed by the diameter of the portal vein. In describing the operation of porta-caval anastomosis, the author states that the portal vein is ligated before the inferior vena cava is dissected. It is however an advantage to free the inferior vena cava before the flow is obstructed by ligation of the portal vein.

In discussing the treatment of ascites the author comes to the conclusion that there is no operative treatment. The pre- and postoperative treatment of the cirrhotic patient is discussed in detail.

At the end of each chapter there are extensive references, and these and others are again given at the end of the monograph. An appendix deals with the current surgical management of portal hypertension in the United States.

Contributors to this appendix are Linton, Sedgwick, Blakemore, Britton, Child, Singleton, Longmire, Madden, Cohn, and McDermott. These authors discuss briefly patients who have survived shunt operations for 5 years, criteria for the selection of patients for a porta-caval and a splenorenal shunt, the management of patients with bleeding oesophageal varices, and the surgical procedures for the correction of ascites. They also mention research being done by them at present in the field of portal hypertension and ascites. This monograph should be read by everyone interested in portal hypertension. The extensive references will be of great value to research workers in this field. B.J.D.

## BONE PHYSIOLOGY AND PATHOLOGY

*Clinical Orthopaedics*. Number 17, Summer 1960. Clinical Physiology and Pathology of Bone. Editor-in-Chief: Anthony F. DePalma, with the assistance of the Associate Editors: The Board of Advisory Editors and The Board of Corresponding Editors. Pp. xi + 387. Illustrated. Single copies: R6.00 net. Subscribers: R4.80 net. London: Pitman Medical Publishing Co. Philadelphia and Montreal: J. B. Lippincott Company. 1961.

This is another book on the physiology of bone, bone-salt, calcium and phosphorus metabolism and metabolic bone disease. Admittedly we have been subjected recently to a spate of rather similar volumes. This one, however, includes a fair amount of very recent work using radioactive isotopes — including osteoclasis studied with yttrium, deposition and removal of radium, use of $Sr^{85}$ in obtaining 'osteograms' in various diseases, bone accretion studies in vitamin-D-resistant rickets, and so on.

The basic sciences are well represented by articles on bone structure, bone crystals, the ivory core of tusks and teeth, the biophysics of human bone, the bone matrix, hyaline cartilage, histochemistry of calcification, nuclei formation, and age changes in bone.

The small amount of 'pure orthopaedics' in this book (e.g. the Stone operation for hallux valgus) seems quite out of place.

On the whole, however, it is informative, interesting and extremely nicely produced. There are many figures and adequate references. Certainly all libraries should possess a copy, also those people especially interested in bone structure or function. Clinical orthopaedists will find it hard going despite the main general title. W.P.U.J.

## MEDICAL EDUCATION

*Medicine: A Lifelong Study*. Proceedings of the Second World Conference on Medical Education. Chicago 1959. Under the Auspices of the World Medical Association. New York: The World Medical Association. London: The British Medical Journal. 1961.

The doctor must be a life-long student of diseases and men and the Second World Conference on Medical Education was concerned with the large proportion of doctors (90% in some areas), who fail to continue their medical studies and reading after graduation. The main reasons for this failure to keep abreast are faulty undergraduate education and the development of faulty habits during that period.

The Proceedings of the Conference, therefore, cover under and postgraduate education, in specialities as well as in general practice, selection and training for research work and the place of research in continuing education. The objective of providing (with the Proceedings of the First World Conference on Medical Education, 1954) 2 fundamental textbooks on medical education has been achieved.

There were 800 delegates from 66 countries at the Conference. 215 contributors are indexed in the book, which is an almost verbatim record of the panel discussions and over 100 papers delivered by renowned authorities. Subjects include preceptorships for students, comprehensive care, hospital work for general practitioners, and group practice. The entire proceedings are summarized in 40 pages by the rapporteurs and vice-presidents.

There is profitable stimulation in this book for those who are concerned about the future of the profession and medical practice, the lot of the general practitioner, and the health and sickness of patients. B.J.K.

## THORACIC SURGERY

*Surgical Diseases of the Chest*. Ed. by Brian Blades, M.D. Pp. 580. 267 illustrations. $22.00. Missouri: The C. V. Mosby Co. Also obtainable from local booksellers. 1961.

This book, which is the combined effort of 18 contributors and dedicated to the memory of Evarts Graham, introduces the student, the practitioner and the surgeon to surgical diseases of the chest. However, thoracic surgery, particularly cardiac surgery, is expanding so rapidly that some techniques and views are outdated even before they are published; it is also inevitable that unnecessary repetition will occur in a book of this kind. Basic physiology and pre- and postoperative care are briefly dealt with, but, surprisingly the value of posture and high-volume suction are not mentioned. The chapters on hiatus hernia and the oesophagus are rather neglected, while ample space is given to the heart and great vessels, and 2 short chapters are devoted to ancillary procedures and anaesthesia for thoracic surgery.

The illustrations enhance the value of this book which deserves the attention of all who are interested in chest surgery. G.S.M.B.

## DENTAL ANAESTHESIA

*Nitrous Oxide in Dentistry, its Danger and Alternatives*. By J. G. Bourne, M.A., M.D. (Cantab.), F.F.A.R.C.S. Pp. x + 181 23 figures. R3.00 net. London: Lloyd-Luke (Medical Books) Ltd. 1960.

The deficiencies of dental anaesthesia prompted the Faculty of Anaesthetists of the Royal College of Surgeons of England to offer a prize of R400.00 for original work in this field. This book represents a welcome contribution.

The author has for years condemned the routine use of nitrous oxide for dental outpatients. After quoting numerous cases of death, permanent brain damage, or delayed recovery following nitrous oxide in the dental chair, he discusses at length its dangers and deficiencies and emphasizes especially the danger of fainting under anaesthesia, which he feels is the major cause of catastrophe.

Following an excellent discussion on inert-gas exchange in the body, nitrous oxide is shown to be unsuitable, while cyclopropane most closely approaches the ideal. Simple apparatus and a readily acquired and simple technique are described with an account of its uses in 3,000 cases.

This small book covers its subject admirably. It is recommended for reading, not only by all doctors and dentists using general anaesthesia for dentistry, but by all anaesthetists. It has a most comprehensive list of references. D.A.U.C.T.

## TOOTH EXTRACTION FOR DOCTORS

*The Extraction of Teeth*. By Geoffrey L. Howe, M.R.C.S. (Eng.), L.R.C.P. (Lond.), F.D.S., R.C.S. (Eng.). Pp. viii + 70. 100 illustrations. R1.75 net. Bristol: John Wright and Sons Ltd. 1961.

Although doctors in South Africa may not undertake dental work if a dentist is available, there are many places in this vast country of ours where they have, of necessity, to attend to the teeth of their patients. This small book will assist such practitioners, since it deals with the basic principles of extractions, usually the only dental work which doctors are called upon to undertake. It is simply written and clearly and adequately illustrated, and, if it is followed, the lot of the general practitioner in outlying areas will be eased. A.H.T.

## MYO-EPITHELIAL CELLS IN SALIVARY-GLAND TUMOURS

*The Identification and the Rôle of the Myoepithelial Cell in Salivary Gland Tumours*. By Erling A. Mylius. Pp. 59. 27 illustrations. R3.40. Norway: Oslo University Press. Obtainable from P. B. Mayer, Box 713, Cape Town. 1960.

This excellent monograph is one of the *Norwegian Monographs on Medical Science* and has also been published as supplement 139 of Acta Pathologica and Microbiologica Scandinavica. The booklet has been very well produced and contains some excellent photomicrographs and electron micrographs. It presents the results of an investigation into the myo-epithelial cells, which are described in great detail.

With the assistance of the electron microscope, tissue cultures and histochemistry, the author has identified neoplastic myo-epithelial cells in most salivary-gland tumours. He concludes that these tumours develop from mature salivary-gland tissue and that, although some seem to be purely epithelial in origin, the majority are partly or exclusively composed of myo-epithelial cells.

This monograph is strongly recommended and marks an important step in our understanding of salivary-gland tumours. D.J. du P.

## SURGICAL DISEASES OF THE PANCREAS

*Surgical Diseases of the Pancreas.* By John M. Howard, M.D., F.A.C.S. and George L. Jordan, JR., M.D., M.S., F.A.C.S. Pp. xviii + 607. 199 illustrations. R14.70. London: Pitman Medical Publishing Co. 1960.

This is a book that can be highly recommended to everyone interested in the pancreas — surgeon, physician and post-graduate student in surgery and medicine. It is well produced, the paper is of high quality, the illustrations are excellent and the numerous tables and diagrams are clear and to the point. It is well written and the reader is carried effortlessly from one paragraph to the next. At the end of each chapter there is a long list of references. The book is written by physicians who have contributed much to a better understanding of diseases of the pancreas, for example E. E. Ellison writes the chapter on ulcerogenic tumours of the pancreas.

The book starts with a historical sketch by Allan O. Whipple, f. 'lowed by excellent chapters on the surgical anatomy of the pancreas and on the applied physiology of the external secretion. The laboratory diagnosis of pancreatic disease is discussed in a separate chapter. A large part of the book deals with acute pancreatitis: its aetiology, pathology, pathological physiology, diagnosis and treatment. There are chapters on special types of pancreatitis, e.g. that associated with gallstones, with alcoholism and with certain operations. Pancreatic calcification, relapsing and chronic pancreatitis, and pancreatitis in unusual forms are discussed separately. There is a wealth of information in all these sections written by Dr. Howard, who refers extensively to the literature and to his own experience. The chapter on hyperinsulism and island-cell tumours of the pancreas is really a collective review — all cases reported up to January 1958 are summarized in tables extending across almost 100 pages.

The latter part of the book deals with pancreatic and periampullary neoplasms, and is written by Dr. Jordan. The operative technique of pancreaticoduodenal resection is described in detail, and the indications, pre- and postoperative care and complications, as well as morbidity and mortality rates, are discussed. The last chapter deals with pancreatic fistulae, but this one and another on pancreatic trauma are not very informative. Ulcerogenic tumours of the pancreas and pancreatic cysts are also discussed.

It was a joy to review this book, which reads so easily and contains much new information. It can be highly recommended to the teacher in surgery and medicine and as an informative reference work on the pancreas. B.D.

# CORRESPONDENCE : BRIEWERUBRIEK

## MALNUTRITION — WHO IS RESPONSIBLE?

*To the Editor:* Your Editorial[1] of 26 August 1961, under the heading 'Malnutrition in Infants and Young Children', rightly draws attention to the appalling toll of children's lives claimed by kwashiorkor and allied conditions in this country. While the figures you quote relate mainly to the Cape Town area, it cannot be emphasized too strongly that malnutrition in young children has a nation-wide distribution, and is certainly not confined to any one Province.

The means of preventing kwashiorkor are well established, but those of us to whom the disease is a daily occurrence must tend to become more and more frustrated at the absence of any decline in incidence, and the continued lack of any concerted effort to find a solution.

The problem is so vast and so widespread that it is quite beyond the means of Provincial or local authorities who have neither the facilities nor the money to launch an effective preventive scheme. In other words, the problem is a national one, which should be tackled at the highest level, in a similar way to poliomyelitis. The fact that kwashiorkor, unlike poliomyelitis, occurs mainly in non-European children surely does not absolve those departments responsible for the health of the community from taking vigorous action without delay.

Admittedly, although it is known how kwashiorkor can be prevented, the implementation of any effective scheme is bristling with difficulties and, to borrow a radio term, demands 'top level' action. We would urge, therefore, that a Government-sponsored national committee be formed, on which all four Provinces are represented, and with a leavening of clinicians actively engaged in treating malnutrition, to explore ways and means of eradicating this national festering sore from our country.

H. L. Wallace
F. Walt
N. M. Mann
Joan Scragg

Department of Paediatrics
University of Natal
9 September 1961

1. Editorial (1961): S. Afr. Med. J., **35**, 705.

## HAND INJURY IN CHILDREN IN RURAL AREAS

*To the Editor:* I should be grateful if you could bring to the notice of medical colleagues what I have found to be a common hand injury of children in our local rural area, the idea being to find out whether any rural practitioners have treated similar injuries recently. This is with a view to lending weight to an attempt to organize a simple preventive measure.

The injury is usually a trivial one, but in one patient it has been crippling and it could be crippling more often.

The injury is caused by the very commonly used machine of the 'mealie-crusher' type. The mealie corns are fed into a large receptacle at the top of this machine and come out, finely ground, through a wide spout at the bottom. This spout is large enough to admit even an adult hand with ease. Apparently, in the machines locally used, there is easy and direct access to the crushing mechanism through the spout. I believe that this is the case with most of these machines. Children, being naturally curious, often put their hands up this spout while mealies are being crushed.

During the past 12 months (almost the entire time I have been in rural hospital practice), I have seen 6 such casualties, the last only a few days ago. The ages of the children ranged from 4 to 7 years. In each of the cases the right hand was involved. In all but one the middle finger was the most seriously affected digit.

In most cases the tip of the distal phalanx was removed, with lacerations of the adjoining fingers. In one patient, aged 5, owing to the extent of the injury, the middle finger had to be amputated. This is the only case in which there will be any permanent disability of a serious nature.

A reason why I suspect this type of injury to be more widespread than these local cases may warrant one to believe, is that 5 adults with whom I am acquainted suffered this type of injury in childhood — victims of the machine and curiosity! One sustained her injury in Ireland, the others in different parts of South Africa. The scars are typical; all on the middle finger of the right hand, all caused by the same type of machine. My senior colleague at this hospital and myself were both similarly injured as children.

It would be interesting to hear of other injuries, if any, caused as those described above.

A simple and cheap preventive measure, in the form of a wide-mesh wire guard or other device, should not be difficult to fix to the machines. It may be that some such device is already in use on newer machines: I will find out! In the meantime, I feel it would be worth while attempting to discover how prevalent this injury still is. I have not been able to discover anything in the literature.

H. F. J. Schumann

Appelsbosch Mission Hospital
P.O. Kingscliffe
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6 September 1961